THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—22ND YEAR. SYDNEY, SATURDAY, AUGUST 31, 1935. No. 9.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.

"Acute Aseptic Meningitis", by I. M. ALLEN, M.D., M.R.C.P., and F. M. SPENCER, M.D., F.R.C.S.E. 275
"Notes on Maternal Mortality", by KENNETH WILSON, M.B., Ch.M., M.C.O.G. 281
"Maternal Mortality: Some Practical Points in Prevention", by M. ELLIOT SMITH, M.B., Ch.M., L.M., F.R.C.S., F.R.A.C.S., M.C.O.G. 285
"The Use of Composite Filters in X Ray Therapy", by C. E. EDDY, D.Sc., F.Inst.P., and JOHN O'SULLIVAN, M.D., B.S., D.M.R. and E. 288

REPORTS OF CASES—

"An Unusual Intracranial Emergency", by GILBERT PHILLIPS, M.Sc., M.B., B.S. 289
"Quinine Amaurosis", by GEORGE SIMPSON, M.B., B.S., M.R.C.P., Dip.G.O., and ARCHIE S. ANDERSON, M.B., B.S., D.O.M.S., F.R.A.C.S. .. 290
"Epidemic Conjunctivitis", by K. S. JONES, M.B., B.S. 291

REVIEWS—

Respiration 291

LEADING ARTICLES

Toxæmia of Pregnancy and Cardio-Vascular Disease 293

CURRENT COMMENT—

Polycythæmia Vera 294
Bacteria and Gastric Ulcer 295

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Ophthalmology 296
Oto-Rhino-Laryngology 297

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 298
Annual Meeting, Melbourne, 1935 299
Nominations and Elections 300

CORRESPONDENCE—

Ethyl Chloride in Anæsthesia 300

OBITUARY—

Dixie Paumier Clement 300
Charles Horsfall Armitage 301

A MASONIC LUNCHEON 302

A WARNING NOTICE 302

DIARY FOR THE MONTH 302

MEDICAL APPOINTMENTS 302

MEDICAL APPOINTMENTS VACANT, ETC. 302

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 302

EDITORIAL NOTICES 302

ACUTE ASEPTIC MENINGITIS.

By I. M. ALLEN, M.D. (New Zealand), M.R.C.P. (London),
Assistant Physician, Wellington Hospital, New Zealand,

AND

F. M. SPENCER, M.D. (New Zealand), F.R.C.S.E.,
Assistant Physician for Diseases of Children, Wellington Hospital, New Zealand.

ACUTE aseptic meningitis is an acute inflammatory reaction of the meninges without organisms in the cerebro-spinal fluid on examination or culture. Cases have been reported during the past thirty-five years, and for ten years have been occurring both sporadically and in epidemics in Europe, Asia and America. In this paper there is summarized a series of cases which, with one exception, occurred in New Zealand.

Case Reports and Commentaries.

Case I.

A girl, aged six years, was seen with Dr. J. R. Harris, of Muswell Hill, on July 22, 1931. The illness began gradually eight days before with severe headache. On the third day the temperature varied from 37·8° to 38·9° C. (100° to 102° F.), the child vomited and complained of pain in the ears; and general hyperæsthesia, rigidity of the back of the neck and a doubtful Kernig sign on the right side were found. On the fifth day the condition of the optic discs was doubtful and the ear drums were normal. On the sixth day the temperature was normal.

The child was bright and talked freely. She was lying on the right side with the head straight, some rigidity of the posterior neck muscles, a pronounced lordotic curve and the legs flexed. The fauces were congested and the upper cervical lymph glands were slightly swollen and tender. Slight nystagmoid jerking appeared on conjugate deviation to the right, the optic disks were swollen and the retinal veins were congested and tortuous. There was slight delay of the right angle of the mouth when talking. The right upper limb drooped when extended with the eyes closed, motor power and muscle tone were decreased, repeated alternating movements were clumsy, and there was uncertainty in approaching the forefinger to an object. The right lower limb was flexed, muscle tone was increased in the

extensors at the ankle, movements were weak, the knee jerk was faint, the ankle jerk was active and the plantar reflex varied from flexor to extensor. The left knee jerk was absent and the left plantar reflex was constantly extensor. The right abdominal reflexes were absent and the left very faint. The spinal muscles were rigid, and the abdomen was pushed forward, distended and tympanitic. Extension of the left lower limb at the knee with the thigh flexed resulted in rigidity of the hamstrings, pain and limitation of movement to 90°. There was a similar but less pronounced finding on the right side. The remainder of the physical examination revealed no abnormality.

Examination of the cerebro-spinal fluid showed that the pressure was raised; a coagulum was present on standing. The total protein content was 0·05%. No excess of globulin was present and glucose and chlorides were within normal limits. Twenty-one cells per cubic millimetre were present and of these twenty were lymphocytes. The Lange colloidal gold curve was normal. No organisms were found on detailed examination. (Dr. J. G. Greenfield.)

Apart from some spasm of the hamstrings which was readily controlled by persuasion there were no physical signs one week later. The child recovered completely two and a half weeks from the onset.

In this case a provisional diagnosis of tuberculous meningitis was made on the sixth day following examination of cerebro-spinal fluid and a guarded prognosis was given. The unusually clear mental condition after an illness of one week suggested that a less serious condition might be present. Otitic hydrocephalus and otogenic meningitis were excluded. The symptoms and physical signs disappeared gradually after drainage of cerebro-spinal fluid and except for the appearance of hysterical contracture of the hamstrings the progress of the patient was uneventful. The clinical findings were in three groups: those of increased intracranial pressure, those of meningeal inflammation, and pains in the lower limbs which appeared to be due to nerve root involvement within the subarachnoid space.

Case II.

A man aged thirty-two years was admitted to hospital on July 21, 1932, on the recommendation of Dr. Gordon Kemp. He complained of frontal and occipital headache of fourteen days' duration, at first constant, later intermittent; of nausea and vomiting after food; of pains in the calves and of difficulty in walking. He had fainted twice and on the day of admission to hospital there had been double vision for ten minutes. He had had mumps and diphtheria in childhood and rheumatic fever ten years before.

He was mentally alert; his temperature was 37·2° C. (99° F.), his pulse rate was 90, and his respiration rate was 20. Transient nystagmus appeared on lateral deviation to the right and left, and anti-clockwise movements on looking up. The optic disks were swollen, the right more than the left, and the retinal veins were engorged. Voluntary movement of the right angle of the mouth was slightly delayed and the uvula deviated to the right on phonation. In the lower limbs clonic movements appeared on exposure, motor power was impaired at the knees, the quadriceps and calf muscles were tender, and sensory and reflex functions were normal. In all other respects the functions of the nervous system were normal. There were a few rhonchi at the bases of the lungs. The urine was acid and contained a few red blood cells and many oxalate crystals. Neither the blood nor the cerebro-spinal fluid yielded the Wassermann reaction. Subsequently the right pupil and palpebral aperture were smaller than the left and after the administration of aspirin the right side of the forehead sweated, whereas the left failed to do so. The cerebro-spinal fluid pressure was normal; the globulin was increased; forty-five lymphocytes per cubic millimetre were present; the figure for examination of chlorides was 500. (Dr. J. O. Mercer.)

For three weeks the evening temperature was 37·2° C. (99° F.). After one week there were no pains in the legs, the swelling of the optic disks was subsiding and the right pupil was smaller than the left. After eleven days there was occipital headache on waking, anti-clockwise movements appeared on deviation of the eyes to the right, the pupils were smaller and the right smaller than the left, and the margins of the optic disks could be defined. Pressure of the cerebro-spinal fluid was normal, globulin was moderately increased, and there were eighteen lymphocytes per cubic millimetre. A fortnight later the pupils were equal and the fluid contained four lymphocytes per cubic millimetre.

In this case the symptoms of increased intracranial pressure were more evident, those of meningeal irritation were minimal, and pains in the lower limbs were a prominent feature of the illness. It was necessary to exclude intracranial complications of renal disease, space-limiting lesions within the cranial cavity and syphilitic disease of the meninges. Epidemic encephalitis came under consideration. The findings were those of slight increase of intracranial pressure, meningeal inflammation, involvement of nerve roots within the subarachnoid space, and involvement of the sympathetic path on the right side probably within the brain stem. The condition was rather more chronic than usual, and there was some evidence of involvement of cerebral tissues within the brain stem in addition to that of meninges and of spinal roots.

Case III.

A man, aged twenty years, was referred by Dr. W. J. Hutchison and admitted to hospital under the care of Dr. Robert Stout with a suspicion of head injury. He could give no history, was irritable, and resented being touched. No abrasions or areas of tenderness were found. The limbs were rigid, the knee and ankle jerks were active and equal, and both plantar reflexes were apparently extensor. X ray examination of the skull revealed no bony abnormality.

When examined four days later he was dull, but cooperated within limits. The eyeballs were restless, the right pupil was larger than the left, and the optic disks were normal. There was delay of the right angle of the mouth when speaking. The left upper limb wandered when extended with the eyes closed. In both upper limbs tremor appeared in the fingers on movement, there was difficulty in handling small objects and the approach of the forefinger to an object with the eyes open was inaccurate. There was slight difficulty in recognizing changes in position of the left toes, the knee and ankle jerks were faint on reinforcement, the right plantar reflex was flexor and the left varied from flexor to extensor. He stood with the feet well apart and swayed backwards and to either side. He walked on a broad base and lurched from side to side. Speech was indistinct and difficult to understand. In other respects the functions of the nervous system and of other systems were normal. The temperature was 37·2° C. (99° F.), pulse rate was 60, and respiration rate 35. The Wassermann test gave no reaction with either the blood or cerebro-spinal fluid.

Examination of the cerebro-spinal fluid on the fourth day showed that the pressure was raised; a great excess of globulin was present, and the lymphocytes numbered 258 per cubic millimetre. (Dr. Mercer.) On the eighth day the pressure was raised; the fluid was straw-coloured, and it contained fibrin clot on standing. The globulin was increased; the fluid contained 417 lymphocytes per cubic millimetre. No tubercle bacilli were present, and inoculation of a guinea-pig did not yield evidence of tuberculosis. On the thirteenth day the pressure was raised; an excess of globulin was present, and 1,280 lymphocytes per cubic millimetre were found. After four weeks the pressure was slightly raised, a slight excess of globulin was present, and 224 lymphocytes per cubic millimetre were found.

During the second week the patient became more alert, the left pupil was still larger than the right, the tendon reflexes were elicited more readily and the temperature, pulse and respiration became normal. He then stated that three days before admission to hospital he had been feverish and suffered from headache. After four weeks headache appeared on two successive mornings and drainage of cerebro-spinal fluid was recommenced. On the thirtieth day there was pain in the shoulders, and the back of the neck was becoming stiff, symptoms which were relieved after further drainage of cerebro-spinal

fluid. During the following four months the only abnormalities were occasional indistinctness of speech and slight headache for two days.

A young man was admitted to hospital in circumstances which suggested the presence of the results of head injury. Subsequent findings pointed to tuberculous meningitis, but detailed analysis of the history and clinical features supported the diagnosis of benign lymphocytic meningitis. The symptoms were controlled after drainage of cerebro-spinal fluid, which showed a higher cell count after the second and third drainages than at the time of the first examination. Complete recovery occurred. The outstanding features were the mental dulling and irritability, the lymphocytic meningeal reaction, evidence of drainage of cells from perivascular lymph spaces, increased intracranial pressure without ocular abnormalities, evidence of irritation of spinal roots within the subarachnoid space, and the reappearance of symptoms when drainage of cerebro-spinal fluid was suspended too early.

Case IV.

A boy, aged nine years, was seen with Dr. J. T. Harding on April 3, 1933. He complained of pains in the head and neck, feverishness and stiffness of the neck for forty hours. Sleep was disturbed by pain and he vomited twice. He had scarlet fever at four, bronchitis for four years after it, and the right pupil was permanently larger than the left.

The child was alert and answered questions well. There was slight tenderness on percussion of the frontal region. Slight nystagmoid movements appeared on lateral deviation of the eyeballs, the optic disks were slightly swollen and the retinal veins were engorged. The head was retracted and the back of the neck was rigid. The left upper limb showed tremor on extension and wandered with the eyes closed. Extension of the knees with the thighs flexed was limited to 90°. In all other respects the nervous system and the other systems were normal. The pressure of the cerebro-spinal fluid was raised, a slight excess of globulin was present and it contained 273 cells per cubic millimetre, most of which were polymorphonuclear cells; no tubercle bacilli or other organisms were found. (Dr. Lynch.) Considerable relief followed drainage of cerebro-spinal fluid. Transient weakness of the left upper limb disappeared after two days. The patient made a rapid recovery.

The condition was characterized by a rapid onset, evidence of meningeal irritation and the rapid development of symptoms of increased intracranial pressure. The cerebro-spinal fluid showed evidence of an acute meningeal reaction, and there was no evidence of the nature of the infective agent. Poliomyelitis came under consideration, but a detailed analysis of the whole of the findings made it improbable.

Case V.

A boy, aged six years, was referred by Dr. T. L. Parr on October 22, 1933. He was recovering from measles and that day had become drowsy and later comatose. On the second day the rash was fading. The temperature was 37·8° C. (100° F.) and the pulse rate was 60. The child was unconscious, the pupils contracted to light, there was slight spasticity of the limbs, and a slight Kernig sign was elicited. In other respects the nervous system and the other systems were normal. The urine contained a trace of albumin. On the second day the pressure of the cerebro-spinal fluid was moderately increased, a great excess of globulin was present, and 150 lymphocytes per cubic millimetre were found. (Dr. Mercer.) On the third day the pressure of the cerebro-spinal fluid was increased, an excess of globulin was present, 68 lymphocytes per cubic millimetre were found, and the chlorides were normal. On the third day the child could be roused, Brudzinski and Kernig signs were present, there was general muscular hypertonia, and the knee jerks could not be elicited. On the fourth day he was clear mentally and muscle tone was normal. On the fifth day there was extreme mental clarity, the head was retracted with some rigidity of the posterior neck muscles, the limbs were hypotonic, and the lower limbs were sensitive to touch. During the next week the child improved steadily, and he recovered completely within two weeks of the onset.

The onset during the active phase of an infectious disease and the lymphocytic picture in the cerebro-spinal fluid suggested tuberculous meningitis. A review of the complete clinical picture led to a diagnosis of benign lymphocytic meningitis complicating measles. The outstanding features were the evidence of meningeal involvement and the rapid and startling interference with consciousness which was controlled only by reducing the intracranial pressure repeatedly to a normal level.

Case VI.

A girl, aged six years, was referred by Dr. J. T. Harding on November 1, 1933. Twenty-four hours before she vomited and became drowsy and feverish. On the day of admission to hospital she was feverish, irritable and delirious. Five years before an elder sister had died with a diagnosis of tuberculous meningitis.

The temperature was 38·9° C. (102° F.), the pulse rate was 120 and the respiration rate 36. The child was drowsy and irritable, tossed about and resented being touched. The posterior neck muscles were rigid, and Brudzinski and Kernig signs were present. The pupils were dilated, the abdominal reflexes could not be elicited and the knee jerks were sluggish. Otherwise the physical examination revealed no abnormality. On the second day the pressure of the cerebro-spinal fluid was raised, and 33 cubic centimetres were removed; it was opalescent. A slight increase in globulin was present, it contained 326 lymphocytes per cubic millimetre; the figure for chloride estimation was 645. (Dr. Mercer.) On the third day 35 cubic centimetres were removed; an excess of globulin was present. It contained more than 0·1% of protein and 268 lymphocytes per cubic millimetre. On the third day the general condition of the child was unchanged and the right parotid gland became swollen. On the fourth day the child talked rationally and the temperature was normal. On the seventh day she was mentally clear and there were no physical signs. On the ninth day some spasm of the posterior neck muscles was evident. The child was discharged on the nineteenth day completely recovered.

The onset with meningeal irritation and the family history of tuberculous meningitis suggested tuberculous meningitis; but a review of all the clinical features twenty-four hours after admission supported the diagnosis of benign meningitis associated with epidemic parotitis and preceding the swelling of the parotid gland. The prominent features were the meningeal reaction, the rapid increase in intracranial pressure, the reaction of the patient with irritability and delirium, and the appearance of meningitis before parotitis.

The Occurrence of Benign Forms of Meningitis and Hydrocephalus.

A condition with signs of increased intracranial pressure and a clear cerebro-spinal fluid on lumbar puncture was recognized by Quincke in 1897 and described as a benign form of meningitis or "serous meningitis". In 1913 Passot classified non-purulent complications of *otitis media*, and later Warrington and Aboulker intracranial serous effusions from all causes into: (i) the so-called serous meningitis with slight symptoms of meningitis and an excess of albumin and cells in the cerebro-spinal fluid, and (ii) an hypertensive condition with symptoms of increased intracranial pressure and a normal cerebro-

spinal fluid. Recently Symonds has considered the hypertensive type with *otitis media* as otitic hydrocephalus.

A benign form of meningitis with increased pressure of the cerebro-spinal fluid and a lymphocytic picture, lymphocytes predominating or polymorphonuclear cells predominating, has attracted attention during the past ten years. Cases have occurred both sporadically and in epidemics. In 1930 Gunther found in the literature from 1898 to 1928 one hundred examples of the condition and suggested the name "aseptic meningitis". Since then at least one hundred more cases have been reported.

In 1910 cases were reported from an epidemic in Paris by Laubry and Foy, and by Rist and Rolland. Guillain and Richet described characteristic cases with jaundice, and Rist pointed out that epidemic jaundice was prevalent in Paris at the time. In 1924 Ustvedt, of Oslo, recognized among 205 cases of meningitis of all types 29 cases of aseptic meningitis, two of which were fatal; and in 1925 Wallgren reported 19 cases from an epidemic in Stockholm. In 1928 cases in children were described from Leipzig by Haessler, and from Dusseldorf by Eckstein. In six of the latter's 12 cases the cerebro-spinal fluid was under increased pressure, and in two cases only was the cell count above normal. Cases of benign lymphocytic meningitis were collected by Danna, Gibbens, Biret, Fournaud and Berkesy. Schneider reported 24 cases from Germany, found the condition occurred chiefly in middle-aged adults, and recognized the occasional appearance of papillœdema. Braithwaite and Innes noted the occurrence during three years of 35 sporadic cases affecting patients whose ages varied from eight months to sixty-four years, and described from an epidemic in children 13 cases, five of which were primary and eight secondary to bronchopneumonia. Rankin reported two cases, in one of which lymphocytes predominated throughout and in the other the cells changed from an excess of polymorphonuclear cells to an excess of lymphocytes. Three cases of aseptic purulent meningitis were described by Micks. Series of cases of aseptic meningitis have also been reported from Japan by Kaneko and Aoki, and from America by Brown and Symmers.

Two of Ustvedt's 29 cases were associated with mumps, one patient had a positive Widal reaction, two had just been vaccinated, and two later developed symptoms resembling those of epidemic encephalitis. Among 12 cases of meningitis and encephalitis with infectious disease reported from Copenhagen by Schiodt there were cases of meningitis with measles, rubeola, parotitis and vaccination.

In 1902 Monod described an increase of lymphocytes in the cerebro-spinal fluid of six out of eight cases of mumps; and in 1917 Massary, Tocmann and Luce observed it in 49 out of 56 cases, in only 16 of which were there symptoms of meningeal involvement. In 1928 Taillens could find in the literature only 37 cases of meningitis associated with mumps, but since then reports of at least 45 others have appeared. Only two of the 37 cases reviewed by Taillens were fatal.

In 1921 Bénard observed in an epidemic of 291 cases of measles 13, or 4·5%, with meningeal complications. These appeared between the sixth and eighth days of the disease and varied from transient symptoms of involvement of the meninges to a condition which was serious and even fatal. Lioret collected ten cases in 1930, found lymphocytes usually predominating in the cerebro-spinal fluid, and regarded the ætiology as uncertain. From an epidemic of measles Morquio described five cases of encephalitis—three generalized, one circumscribed with hemiplegia, and one affecting the pons and medulla; one case of diffuse myelitis; and two cases of meningitis—one resembling tuberculous meningitis and the other resembling a radicular and polyneuritic syndrome. In 1926 Anderson illustrated the possible gravity of aseptic meningitis complicating measles in her report of a case in which the onset was rapid, in which coma supervened within twenty-four hours, and in which recovery followed repeated lumbar puncture.

Rarer accompaniments of aseptic meningitis have been malaria reported by Porot, paratyphoid fever with aseptic purulent meningitis by Lesne, typhoid fever by Laroche and Peju, anti-typhoid, paratyphoid *A* and *B* inoculation by Roussy and Cornil, diagnostic lumbar puncture by Reynolds and Wilson, and diphtheria of which Kapetis collected 19 cases. De Lauvergne and Abel described a meningeal reaction in the course of serum sickness, and cases have been described following head injury.

Zoster meningitis is characterized by an excess of lymphocytes and a raised protein content in the cerebro-spinal fluid. In some cases there may be definite clinical evidence of meningitis, in others there is gross involvement of the spinal cord with the clinical picture of zoster meningo-myelitis, and rarely the substance of the brain is involved. With varicella involvement of the brain and spinal cord is more common than involvement of the meninges alone. Of 32 cases of neural complications of varicella collected from the literature by Brain, in only two cases was the stress on the meninges; and among 37 cases collected by Fasella six patients suffered from "serous meningitis" and two from meningo-myelitis.

The Clinical Features of Acute Aseptic Meningitis.

The onset is apparently acute, though in many cases there have been a few days of general malaise before the onset of the acute phase. The patient becomes very ill and in some cases appears to be in danger of his life. The duration of the illness varies from three days to four weeks, and averages from seven to fourteen days. With correct treatment the progress towards recovery is uniform; otherwise the clinical course may be intermittent, show steady deterioration and even end fatally. Patients in whom there is a rapid increase in intracranial pressure are definitely in danger. Sequelæ are uncommon.

The acute phase begins suddenly with severe headache, vomiting and pains in the neck and limbs. Headache is usually occipital, but may be frontal and vertical. Fever is high and subsides by lysis or crisis; in a few cases fever is intermittent, and some cases are afebrile. Pains in the limbs are severe and muscles in the areas affected may be tender. Usually the patient is conscious, alert and

mentally clear. Occasionally he is restless and irritable. In a few cases there are lethargy and mental confusion, delirium may be observed and intractable insomnia has been noted by Gibbens. With rapid increase in intracranial pressure the patient becomes apathetic and drowsy, and may be comatose within twenty-four hours of the onset of the condition. In infants the anterior fontanelle bulges. Occasionally a slight squint is observed, and, when pressure has risen rapidly, nystagmoid jerkings of the eyeballs may appear on lateral deviation. Photophobia is apparent in some cases. Occasionally the pupils are unequal in size and contract slowly on exposure to direct light. In some cases a slight degree of papillœdema is observed, and Eckstein has reported hæmorrhage into the fundus. In the older children of Braithwaite's and Innes's series there was no papillœdema; but it has been seen by Gibbens, Eckstein, Schneider and in the present series. Facial palsy has been seen occasionally. The posterior neck muscles may be rigid and tender, and sometimes the muscles of the back are similarly affected. The degree of rigidity varies with the rate of onset and the extent of meningeal involvement. Forcible flexion of the head may produce flexion of the upper limbs, and extension of the knee with the thigh flexed may be impossible beyond 90°. With mental alertness the tendon reflexes are usually over active and the plantar reflexes flexor; but when pressure has risen rapidly it may be difficult or impossible to elicit the tendon reflexes, and one or both of the plantar reflexes may be extensor. With involvement of nervous tissues there may be weakness of one or more limbs of an upper motor neurone type with impairment of muscle tone and diminution of tendon reflexes.

The cerebro-spinal fluid is usually under increased pressure, often moderate, but sometimes very high. In Gordon's and Abrahams's case and in Case III the pressure was normal. The fluid is either cloudy or clear; sometimes it is purulent. In Gordon's and Abrahams's case it was opalescent on the sixth day. In Case III in this series it was straw-coloured, and in Verillotte's and Le Blaye's case it was yellow. In three cases of Eckstein's series and in Cases I and III of this series a cobweb clot appeared on standing. The cell count varies in most cases from 60 to 1,500. In ten of Braithwaite's and Innes's cases the cell count was under five; and in Feiling's case of mumps meningitis it was 2,500. In most cases the cells are lymphocytes when the patient comes under observation, but in some cases at the outset polymorphonuclear cells may predominate. In mumps and measles meningitis the cells are chiefly lymphocytes. In 27 of Andersen's and Wulff's cases lymphocytes predominated or were in excess, in two there were equal numbers of lymphocytes and polymorphonuclear cells, and in three cases polymorphonuclear cells predonimated. As shown by Schiff, the change from a polymorphonuclear to a lymphocytic picture may be rapid. In Rankin's case there were 300 cells per cubic millimetre, and the percentage of polymorphonuclear cells fell from 96 on the fifth day to 30 on the seventh day and 20 on the ninth day. In Gordon's and Abrahams's case there were 280 cells per cubic millimetre on the sixth day—40% polymorphonuclear cells, 30% small lymphocytes, and 30% large endothelial cells; on the seventeenth day there were 250 cells and 90% lymphocytes; and on the twenty-third day there were eight lymphocytes. The total protein is increased, and glucose and chlorides remain within normal limits. Occasionally the former may be slightly increased and the latter slightly decreased. No organisms are found in the cerebro-spinal fluid; search for tubercle bacilli, even by the most refined methods, is negative; and inoculation of a guinea-pig is without result.

Associated conditions have been: broncho-pneumonia in some of Braithwaite's and Innes's cases; congestion of the throat and enlargement of the cervical lymph glands in Case I; and sore throat from which a hæmolytic streptococcus was grown in the majority of Schneider's cases. In Case II crepitations appeared at the bases of the lungs, and a few red blood cells were found in the urine.

The Nature of Idiopathic Acute Aseptic Meningitis.

There is little direct knowledge of the pathology. Eckstein injected cerebro-spinal fluid from cases of aseptic meningitis suboccipitally into three apes. Three days later one of them developed fever and ataxia, at first the cerebro-spinal fluid showed an excess of polymorphonuclear cells and later of lymphocytes, and the fluid was sterile throughout. Eighteen days later severe histological changes were found—perivascular infiltration, necrosis and glial infiltration of the medulla and mid-brain. Taillens quotes the findings of Acker in a case of mumps meningitis. These included congestion of the convexity of the brain, hydrocephalus, layers of fibrin at the base and a complete absence of changes in the spinal cord.

A possible relationship with cerebro-spinal meningitis, poliomyelitis, tuberculous meningitis or epidemic encephalitis has been reviewed by Gibbens, and more recently by Abrahamson.

It has been suggested by Berkesy that the condition represents an abortive form of poliomyelitis. Gunther observed a tendency for cases to occur in the autumn. Wallgren drew attention to the transition from polymorphonuclear cells to lymphocytes in the cerebro-spinal fluid, and Gibbens quotes Antoni as having observed a family in which one child suffered from poliomyelitis and another showed an aseptic meningeal reaction. On the other hand, in none of the epidemic series, except those reported by Berkesy and Gunther, were the cases associated with the simultaneous occurrence of poliomyelitis in the country. No case of flaccid paralysis of lower motor neurone type has occurred after an attack of aseptic meningitis. The complete clinical picture is distinct from that of poliomyelitis; the excessive meningeal reaction and the rapid rise in intracranial pressure are characteristic features of many cases of aseptic meningitis; and in its essential features the clinical picture of aseptic meningitis presents a uniformity which is unknown with poliomyelitis, even during a single epidemic.

Tuberculous meningitis is the condition diagnosed in many cases. Tubercle bacilli are never found in the cerebro-spinal fluid and inoculation of a guinea-pig is also "negative". Wallgren considered a relationship in one case on the finding of a calcified mediastinal gland on X ray examination; and Andersen and Wulff drew attention to a case with a positive von Pirquet reaction, in which six years later there was evidence suggesting the presence of calcified tuberculomata in the brain.

Epidemics of aseptic meningitis and epidemic encephalitis do not coincide; and an active meningeal reaction does not occur with the more orthodox types of epidemic encephalitis. On the other hand, two of Andersen's and Wulff's patients with a characteristic picture of aseptic meningitis developed later physical signs pointing to involvement of the same areas of the brain stem as are affected in epidemic encephalitis. There are in the literature five other cases in which the same sequence was observed and, as suggested by Abrahamson, it is not improbable that the idiopathic cases represent a hitherto unrecognized clinical form of epidemic encephalitis with a good prognosis.

At first the epidemic meningo-encephalitis which occurred in St. Louis in 1933 and was associated with a pronounced meningeal reaction suggested a relationship of this type, but more recent investigation of the material by Kinsella and Brown has shown that it is probably due to a virus which differs from that associated with a previous Japanese epidemic, and bears no relation to epidemic encephalitis.

There are now on record five families in which some members have been affected by parotitis, some by lymphocytic meningitis alone and some by both conditions. In a family mentioned by Wallgren one child had aseptic meningitis and another bilateral parotitis. In another family described by Paddock four children were affected. One child developed parotitis; three weeks later two others were affected, one with lymphocytic meningitis and the other with parotitis and meningeal symptoms; and during their convalescence a fourth child developed parotitis. Wollstein produced aseptic meningitis by injecting saliva from children with mumps into the subarachnoid space of cats. The cerebro-spinal fluid was under increased pressure, the total protein was increased, and 66% to 84% of the cells were polymorphonuclear. The experimental work of Gordon supports the presence of a common agent in the production of mumps and the meningitis associated with it.

Sheldon has described a case in which the findings suggested an allergic basis. The meningeal reactions observed in the course of serum sickness by de Lauvergne and Abel were undoubtedly an accompaniment of the serum sickness and not an independent condition; and Greenfield has recently drawn attention to allergy as a possible factor in the production of aseptic meningitis with infectious fevers.

Observations available support the recognition of acute aseptic meningitis as a clinical entity, but there is no evidence to show that all cases are the result of a common factor. Mumps meningitis appears to be an essential part of the disease, zoster meningitis is a usual accompaniment of zoster, and with varicella the evidence is almost equally balanced between the agent associated with the disease and an independent agent. The allergic cases stand alone. For the rest, as with encephalitis, the evidence is rather in favour of an independent agent which either operates alone or is activated by the preceding or accompanying disease. The activation of a neurotropic virus by another infection in the louping-ill of sheep supports this view. From the limited experimental work, the occurrence of signs of tissue damage in some cases of aseptic meningitis and the neurological complications of individual epidemics of infectious disease, it is suggested that these cases represent the extreme of a sequence which includes aseptic meningitis and meningo-encephalitis, encephalomyelitis, generalized encephalitis and local encephalitis, occurring in both an idiopathic form and as a sequel of infectious fevers, the clinical picture varying with the relative stress of the infection on the meninges and different parts of the nervous tissues.

Problems in Differential Diagnosis.

The cases described in this paper have drawn attention to most of the problems in differential diagnosis. Of conditions which produce a rapid increase in intracranial pressure, supratentorial tumour with rapidly developing cerebral œdema, and subtentorial tumour with secondary hydrocephalus may come under attention with symptoms and physical signs resembling those of a meningeal reaction. Intracranial complications of middle ear disease, including aseptic meningitis as a neighbourhood effect, aseptic meningitis associated with cerebral abscess, and otitic hydrocephalus may cause difficulty. Careful review of all the clinical features is essential as a guide to the risk and wisdom of withdrawing cerebro-spinal fluid.

Tuberculous meningitis is so often diagnosed that it is necessary to adopt a restrained attitude towards such a possibility, so that the patient with a clinical picture resembling superficially that of tuberculous meningitis may receive the treatment necessary for his recovery. Aseptic meningitis associated with other conditions is usually diagnosed with ease when the other condition has preceded the onset of the meningeal reaction, but is apt to be misinterpreted when the characteristic features follow it. The relative stress on the meninges, spinal roots and tissues of the brain and spinal cord may lead to confusion with other inflammatory conditions of the central nervous system, meningo-radiculitis and acute infective polyneuritis. This difficulty can be overcome only by adequate clinical study of the patient.

Summary and Conclusions.

1. Six cases of acute aseptic meningitis are reported—four idiopathic, one with measles and one with mumps; and attention is drawn to the difficulties in diagnosis which arose in each case.

2. The clinical features resolve themselves into four groups: those of infection, increased intracranial pressure, involvement of the meninges, and involvement of the central and peripheral nervous tissues.

These are present in varying degrees in individual cases, thus producing variations in the average clinical picture of the condition.

3. The occurrence of benign forms of meningitis and hydrocephalus is discussed, and attention is drawn to important features and accompaniments of series of epidemic and sporadic cases.

4. The clinical features are summarized and attention is drawn to the pathology of the cerebro-spinal fluid as a response to an inflammatory reaction in varying degrees at different times.

5. The nature of idiopathic acute aseptic meningitis is discussed and the conclusion is reached that the majority of the cases probably represent a disease *sui generis*, due to an agent which may operate alone or when activated by other conditions.

6. Cases diagnosed as, or suspected to be, tuberculous meningitis without the presence of tubercle bacilli in the cerebro-spinal fluid should come under careful review.

7. The main points in differential diagnosis are discussed and attention is directed to the dangers of inadequate clinical study of the patient.

8. The diagnosis of aseptic meningitis presumes no definite knowledge of the ætiology of the condition and leaves open the ultimate elucidation of its nature.

Bibliography.

J. L. Abrahamson: "Acute Lymphocytic Meningitis", *Archives of Neurology and Psychiatry*, Volume XXXI, June, 1934, page 1235.

S. Andersen and F. Wulff: "*Meningitis Acuta Serosa Idiopatica Benigna*", *Acta pathologica et microbiologica Scandinavica*, 1930, Supplement 3, page 21.

D. M. Anderson: "Serous Meningitis; Repeated Lumbar Puncture; Recovery", *The British Medical Journal*, June 5, 1926, page 943.

L. Berkesy: "*Ueber Fälle von epidemisch auftretender Meningitis serosa in Sommer 1931 in Szeged*", *Weiner Klinische Wochenschrift*, Volume I, 1931, page 350.

C. Biret: "Acute Benign Lymphocytic Meningitis", *Thèse de Paris*, 1931. Abstracted in *The British Medical Journal*, September 26, 1931, "Epitome of Current Medical Literature", page 47.

W. R. Brain: "Lecture on Zoster, Varicella and Encephalitis", *The British Medical Journal*, January 17, 1931, page 81.

J. V. C. Braithwaite and W. M. Innes: "Epidemic Meningitis Minor", *The British Medical Journal*, September 26, 1931, page 567.

V. Danna: *Thèse de Paris*, 1930.

V. de Lavergne and E. Abel: "*Des modifications du liquide céphalo-rachidien au cours des réactions sériques*", *Bulletins et Mémoires de la Société de Médicine des Hôpitaux de Paris*, Volume I, 1926, page 488.

E. De Massary, Tockmann and Luce: "*Méningite lymphocytique et syndromes nerveux dans les oreillons*", *Bulletins et Mémoires de la Société de Médicine des Hôpitaux de Paris*, Volume XLI, 1917, page 847.

A. Eckstein: "*Epidemische Meningitis Serosa*", *Zeitschrift für Kinderheilkunde*, Volume I, 1931, page 564. Abstracted in *Klinische Wochenschrift*, Volume X, 1931, page 22.

F. Fasella: "Nervous Complications of Varicella", *Il Policlinico (sézione medica)*, Volume XXXVI, November 1, 1929, page 566. Abstracted in *The British Medical Journal*, January 25, 1930, "Epitome of Current Medical Literature", page 15.

A. Felling: "The Blood and the Cerebrospinal Fluid in Mumps", *The Lancet*, July 12, 1913, page 71.

J. Fournaud: "Acute Benign Lymphocytic Meningitis", *Thèse de Paris*, 1931. Abstracted in *The British Medical Journal*, August 8, 1931, "Epitome of Current Medical Literature", page 19.

H. Gibbens: "Acute Aseptic Meningitis", *The Lancet*, July 4, 1931, page 12.

I. Gordon and A. Abrahams: "Case of Acute Aseptic Meningitis", *The Lancet*, October 24, 1931, page 903.

J. G. Greenfield: "Discussion on Nervous Complications of Acute Fevers and Exanthemata", *Proceedings of the Royal Society of Medicine*, Volume XXVII, August, 1934, page 1428.

G. Guillain and Ch. Richet: *Bulletins et Mémoires de la Société de Médicine des Hôpitaux de Paris*, Volume II, 1910, page 127.

A. Gunther: "*Ueber akute 'aseptische' Meningitis*", *Jahrbuch für Kinderheilkunde*, Volume CXXVIII, 1930, page 127.

A. Kapetis: "*Contribution à l'étude des reactions méningées dans les paralysies diphthériques*", *Thèse de Paris*, 1921.

R. A. Kinsella and G. O. Brown: "Clinical Features of Epidemic (St. Louis) Encephalitis", *The Journal of the American Medical Association*, Volume CIII, August 8, 1934, page 462.

G. Laroche and G. Peju: "Typhoid Meningitis", *Bulletins et Mémoires de la Société de Médicine des Hôpitaux de Paris*, Volume I, 1920, page 211.

Ch. Laubry and G. Foy: *Bulletins et Mémoires de la Société de Médicine de Hôpitaux de Paris*, Volume II, 1910, page 236.

E. Lesne: "Paratyphoid B. Suppurative Meningitis", *Bulletins et Mémoires de la Société de Médicine des Hôpitaux de Paris*, Volume II, 1920, page 1162. Abstracted in *The British Medical Journal*, November 27, 1920, "Epitome of Current Medical Literature", page 76.

M. Lioret: "Measles Meningitis", *Thèse de Paris*, 1930. Abstracted in *The British Medical Journal*, April 25, 1931, "Epitome of Current Medical Literature", page 77.

R. H. Micks: "Atypical Meningitis", Section of Medicine of the Royal Academy of Medicine in Ireland, May 23, 1930, *The British Medical Journal*, June 28, 1930, page 1177.

R. Monod: *Thèse de Paris*, 1902.

L. Morquio: "*La méningo-encephalite après rougeole*", *Archives de Médicine des Enfants*, Volume XXXIV, May, 1931, page 269.

B. W. Paddock: "Mumps Associated with Meningitis in Identical Twins", *American Journal of Diseases of Children*, Volume XLIV, September, 1932, page 565.

A. Porot: *Lyon Médical*, Volume CXXIX, 1920, page 389.

A. L. K. Rankin: "Acute Aseptic Meningitis", *The British Medical Journal*, January 23, 1932, page 138.

K. E. Reynolds and G. Wilson: "Aseptic Meningitis Following Diagnostic Lumbar Puncture: Indications for Lumbar Puncture and Complications Secondary to it", *The Journal of the Americal Medical Association*, Volume CII, May 5, 1934, page 1460.

E. Rist: *Bulletins et Mémoires de la Société de Médicine des Hôpitaux de Paris*, Volume II, 1910, page 279.

E. Rist and J. Roliand: "*Méningites benignes d'allure epidémique*", *Bulletins et Mémoires de la Société de Médicine des Hôpitaux de Paris*, Volume II, 1910, page 245.

G. Roussy and L. Cornil: *Revue Neurologique*, Volume V, 1919, page 453.

E. Schiff: "*Ueber gutartige aseptische eitrige Meningitis im Kindesalter*" *Monatsschrift für Kinderheilkunde*, Volume XLVIII, 1930, page 434.

E. Schiödt: "Encephalitis and Meningitis Following Acute Infectious Disease", *Ugeskrift for Laeger*, Volume XCIII, 1931, page 995. Abstracted in *The British Medical Journal*, December 26, 1931, "Epitome of Current Medical Literature", page 111.

H. Schneider: "*Ueber epidemische akute 'Meningitis serosa'*", *Weiner Klinische Wochenschrift*, Volume I, 1931, page 350.

J. H. Sheldon: "Serous Meningitis of Allergic Nature", *The Lancet*, April 15, 1933, page 798.

C. P. Symonds: "Otitic Hydrocephalus", *Brain*, Volume LIV, April, 1931, page 55.

J. Taillens: "Meningitis in Mumps", *Révue médicale de la Suisse Romande*, Volume XLVIII, 1928, page 420. Abstracted in *The British Medical Journal*, July 7, 1928, "Epitome of Current Medical Literature", page 1.

Y. Ustvedt: "Benign Meningitis", *Tidsskrift for de Norske Laegerforening*, Volume XLIV, October 1, 1924, page 877. Abstracted in *The British Medical Journal*, November 29, 1924, "Epitome of Current Medical Literature", page 77.

A. Wallgren: "*Une nouvelle maladie infectieuse du système nerveux central*" *Acta Pœdiatrica*, Volume IV, 1924, page 158.

M. Wollstein: "Experimental Mumps", *Journal of Experimental Medicine*, Volume XXXIV, 1921, page 537.

NOTES ON MATERNAL MORTALITY.[1]

By KENNETH WILSON, M.B., Ch.M. (Sydney), M.C.O.G.,

Honorary Obstetrician, Lady Bowen Hospital, Brisbane; Honorary Out-Patient Gynæcologist, Brisbane Hospital.

WHEN I was asked to read a paper on the subject of maternal mortality, it was suggested that statistical tables be drawn up and presented, which would be comparable with those given in the Anne MacKenzie Oration on March 4, 1935, by Dame Constance E. D'Arcy and published in THE MEDICAL JOURNAL OF AUSTRALIA on March 30, 1935. The Registrar-General's Department was consulted with this end in view. It was found that statistical information with regard to maternal mortality in the State of Queensland, comparable with that given in the oration mentioned, could not be obtained, with the exception of one table. The exception is Table VIII in the oration, and it deals with maternal mortality in New South Wales during the years 1927 to 1933.

It was decided, therefore, to prepare a similar table for the State of Queensland and to compare and discuss the tables in detail. I have prepared in addition a similar table from the records of the Obstetrical Section of the Queensland Branch of the British Medical Association, which, although it deals with much smaller numbers, is yet worth presenting, as it is an exact record of the deaths which occurred.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on July 5, 1935.

It may be said at the outset that it is extremely unlikely that a state will ever be reached at which every pregnant woman can be successfully delivered without a fatality. This does not imply that efforts to reduce the maternal death rate should not be made. It is in fact certain that if the efforts of every organization, institution and individual concerned in the process of childbirth were stimulated to this end, the maternal death rate would be appreciably lessened.

In analysing the tables an attempt will be made to indicate those deaths which were preventible and those which were inevitable. This cannot be done with exactness, except in the table from the Obstetrical Section, as sufficient details are not available; but they are exact enough to serve as a basis for discussion.

The first table to be presented (Table I) deals with maternal mortality in the State of Queensland for the years 1927 to 1933.

TABLE I.
Maternal Mortality in Queensland.

Cause of Death.	Deaths, 1927 to 1933.	
	Number.	Rate per 1,000 Births.
Accidents of pregnancy	32	0·25
Puerperal hæmorrhage	96	0·74
Puerperal septicæmia	91	0·70
Albuminuria and eclampsia	127	0·98
Phlegmasia alba dolens, sudden death, embolism	21	0·16
Other casualties of childbirth, including abortion	294	2·27
Total	661	5·10
Exclude abortions	107	0·83
Amended total	554	4·27

You will notice that the total number of deaths over this period is 661, which is a death rate of 5·10 per 1,000 births, and that included in this figure are 107 deaths from abortion. If these are excluded, the death rate drops to 4·27 per 1,000 births.

In the table you will see that the largest number of maternal deaths, 294, a rate of 2·27, is in the group called other casualties of childbirth, including abortion. It is interesting to compare this figure with that of New South Wales (see Table III), where the rate is 1·45. If abortions are excluded from both sets of figures, other casualties of childbirth become responsible for a rate per thousand of 0·57 in New South Wales and of 1·44 in Queensland. The rate per thousand for the excluded abortions is 0·88 for New South Wales and 0·83 for Queensland.

This group has been found capable of further detailed analysis, and I have drawn up a table to show the conditions contained in it.

A detailed consideration of this table will be of value.

For the purposes of this paper the deaths from abortion will not be discussed, as the prevention of deaths from this cause is a different problem to the prevention of those from obstetrical conditions.

TABLE II.
Maternal Mortality in Queensland, 1927 to 1933. Analysis of Other Casualties of Childbirth, including Abortion.

Cause of Death.	Number.
Abortion	107
Ectopic	43
Other toxæmias	18
Cæsarean section, other surgical operations, other obstetrical procedures	114
Unspecified puerperal conditions: (*a*) disease (puerperal) of the breast, (*b*) others	12
Total	294

With regard to the 43 deaths from ectopic gestation, I should say that under existing conditions this number is stationary. A reduction might be effected if medical advice were sought earlier and if earlier operation were afforded to the patient.

The 18 deaths from other toxæmias include such conditions as *chorea gravidarum*, *hyperemesis gravidarum* and vomiting of pregnancy. The prevention of these deaths depends on the judgement of the practitioner and is the responsibility of the medical profession.

The 114 deaths from Cæsarean section *et cetera* include deaths from such conditions as Cæsarean section, instrumental delivery, craniotomy, breech deliveries and obstetric operations generally. The prevention of deaths in this group also appears to be the responsibility of the medical profession. A diminution in the number of deaths could be expected from improved teaching in medical schools, an increase in the number of recent graduates holding resident obstetric hospital appointments, increased and improved antenatal care, and regular attendance of medical practitioners at post-graduate courses and lectures. The group will never be completely abolished, as none of us, however skilful and experienced, is infallible, and a few at least of our operative procedures will be attended with fatalities.

The last group in the table shows twelve deaths from unspecified puerperal conditions, and included in the group are such conditions as puerperal insanity, abscess of the breast and mastitis. Without access to the actual records of the fatal illness of these patients it is difficult to realize why the number is as large as this, for abscess of the breast and mastitis are not generally regarded as fatal conditions. The obvious preventive measure is earlier contact between the patient and the medical practitioner.

The largest group but one in Table I is that of albuminuria and eclampsia, which contains 127 deaths, the rate per thousand being 0·98. The table for New South Wales shows the rate per thousand in that State to be 1·24. I have for a long time been under the impression that the incidence of albuminuria and eclampsia was much higher in Brisbane than in the southern cities, but if the death rate is any indication of the relative incidence of the conditions, then this opinion is shown to be erroneous by a comparison of the rates for the two States.

TABLE III.

Maternal Mortality Rates per Thousand Births in New South Wales and Queensland Compared, 1927 to 1933.

Cause of Death.	Rate per 1,000 Births.	
	New South Wales.	Queensland.
Accidents of pregnancy	0·53	0·25
Puerperal hæmorrhage	0·76	0·74
Puerperal septicæmia, post-abortive septicæmia	1·51	0·70
Albuminuria and eclampsia	1·24	0·98
Phlegmasia alba dolens, embolism, sudden death	0·39	0·16
Other casualties of childbirth	0·57	1·44
Total (excluding abortions)	5·00	4·27
Abortions	0·88	0·83
Grand total	5·88	5·10

The group includes albuminuria of pregnancy, eclampsia, nephritis of pregnancy, pyelitis of pregnancy and toxæmia of pregnancy.

An appreciable diminution in the number of deaths in this group would be expected from an increase in the number of pregnant women receiving efficient antenatal care. Some deaths would occur in spite of this, as some patients develop eclampsia with very little warning, and there will always be pregnancy among patients who suffer from chronic nephritis, latent or recognized.

The next group for consideration is that of puerperal hæmorrhage, in which the number of deaths is 96. Included under this title are accidental hæmorrhage, *placenta prævia* and *post partum* hæmorrhage. The mortality rate per thousand births for Queensland is 0·74, while over the same period for New South Wales it is 0·76. As one would expect from conditions of this nature, the rate is approximately the same, and it serves to indicate that under existing conditions of medical practice improvement is not to be expected. This group includes some of the most serious obstetrical conditions, and a lessening of the rate must depend partly on the skill of the practitioner and partly on the patient's surroundings, for, if it were possible to treat all these patients in first rate hospitals, then some diminution in the rate might be obtained.

The next group to be considered is that of puerperal septicæmia, and in this group there were 91 deaths, which give a rate of 0·70 per 1,000 births. The group includes post-abortive sepsis, *post partum* pyæmia and puerperal sapræmia and septicæmia. The corresponding group for New South Wales showed deaths from post-abortive septicæmia separately. I have not been able to do this for Queensland, and in order to produce a New South Wales group comparable with that of Queensland I have combined the two divisions in the table for New South Wales. The mortality rate per thousand births for the latter State is 1·51, which is more than twice that of Queensland.

It is not altogether easy to account for a difference as great as this, although a higher rate is to be expected in a State more highly industrialized. The factor difficult of explanation might be found in the certification of death.

The prevention of puerperal septicæmia has been discussed frequently enough of late for me to say only that the condition is largely avoidable and that improvement in the aseptic and antiseptic technique of those engaged in obstetric practice should considerably reduce the death rate from this cause.

The next group is that classified under accidents of pregnancy. The number of deaths in this group was 32, a rate of 0·25 per 1,000 births. This group embraces hydramnios, retroversion of the pregnant uterus, hydatid mole and multiple pregnancy. You will notice that the mortality rate for New South Wales over the same period was 0·53 per 1,000 births. As in the preceding group, the rate for New South Wales is more than double that for Queensland. It is difficult to imagine why this is so, unless the explanation is offered that the classification or notification of deaths is responsible, for there is no reason why any of the conditions included in the group should be less fatal in Queensland than in New South Wales. If a diminution in the number of deaths in this group is to be obtained, then it will be by greater skill and experience of medical practitioners.

The last group in this table for consideration is that under the heading *phlegmasia alba dolens*, embolism and sudden death. There were 21 deaths over the period, which produce a rate of 0·16 per 1,000 births. Included in the group are such conditions as pulmonary embolism, sudden death from cardiac embolism and thrombosis, and death from shock after delivery. In New South Wales for the same period the rate per 1,000 births was 0·39. Here again there is no obvious explanation for the greater number, as the incidence of such conditions as these should be approximately the same. As a group, a lessening of the number is hardly to be expected, but in the case of *phlegmasia alba dolens* and shock improved obstetric practice might diminish the number.

By permission of the Obstetrical Section of the Queensland Branch of the British Medical Association, I have been able to compile some figures, the accuracy of which cannot be in doubt, as every confinement reported, including the maternal deaths, is fully described and contains all the information necessary to make the cause of death certain. Complete records of 3,128 confinements conducted in the private practice of medical practitioners are in the possession of the section. It is necessary to mention that the section receives reports only of pregnant women whose pregnancy has progressed to such an extent that the child is viable, so that abortions and miscarriages are not included in the reports. Of the 3,128 patients, 10 were reported as having died, which produces a death rate of 3·2 per 1,000 births.

A table comparable with Table I is given (Table IV).

TABLE IV.

Maternal Mortality in 3,128 Confinements of the Obstetrical Section.

Cause of Death.	Deaths from 1928 to 1935.	
	Number.	Rate per 1,000.
Accidents of pregnancy	0	0·00
Puerperal hæmorrhage	3	0·96
Puerperal septicæmia	4	1·28
Albuminuria and eclampsia	2	0·64
Phlegmasia alba dolens, embolism, sudden death	0	0·00
Other casualties of childbirth	0	0·00
Acute yellow atrophy of the liver	1	0·32
Total	10	3·2

The death from acute yellow atrophy of the liver has been given a separate group, as in the statistical tables used in compiling the groups deaths from this cause are not included under deaths associated with childbirth.

You will notice that the deaths are from the years 1928 to 1935, which, although not strictly comparable with Tables I and III, are yet sufficiently close to justify the comparison.

A detailed study of the causes of death will be given.

The largest group is that of puerperal septicæmia, which accounts for nearly half the deaths. An abbreviated summary of the four deaths is given.

1. Pelvic cellulitis occurred after a natural delivery. The patient was a *primipara*. Operation of abdominal drainage of cellulitic abscess was performed. The patient died on the tenth day.

2. Natural delivery occurred. An adherent placenta was manually removed. Puerperal septicæmia occurred and the patient died on the thirteenth day.

3. Prolapse of the cord occurred during labour. The cord was replaced. The child was delivered by forceps in the occipito-posterior position. Septicæmia supervened and the patient died in the third week.

4. Natural delivery occurred. Manual removal of the placenta was followed by death from puerperal septicæmia.

In considering these four deaths it is interesting to note that three of the patients were delivered naturally. The first one developed sepsis with the minimum of interference. The second and fourth deaths followed manual removal of the placenta, which is always a dangerous procedure but which at times, in the interest of the mother, must be done. The third death followed more intravaginal manipulations than are usually necessary. The principle that all confinements should be conducted with a minimum of interference is strengthened by the result to this patient.

The next group for consideration is that of puerperal hæmorrhage, and there were three deaths from this cause. Details are given.

5. The patient suffered from concealed accidental hæmorrhage of a severe type accompanied by anuria.

6. *Post partum* hæmorrhage occurred in a debilitated woman.

7. Accidental hæmorrhage of a severe type occurred.

It is worthy of note that in the whole series of reported cases there was only one death from *post partum* hæmorrhage, and from the history it is probable that had the patient not been suffering from an obscure condition causing severe debilitation, the blood loss would not have been sufficient to cause death. The two deaths from accidental hæmorrhage were both of the severe type, and it is doubtful if any means not used could have prevented a fatal issue.

The third group represents two deaths from eclampsia. The details are given.

8. The patient was delivered by low forceps application. Eclampsia occurred after delivery and the patient died on the thirteenth day. Antenatal care was practised.

9. The patient had eclamptic fits followed by acute yellow atrophy of the liver. The condition was confirmed *post mortem*.

From the detailed histories of these patients it is doubtful whether any treatment other than that afforded to them could have altered the result.

There was one patient (number 10) who developed acute yellow atrophy of the liver without albuminuria and who died seventy-two hours after delivery. The diagnosis was also confirmed *post mortem*. It would appear, then, that acute yellow atrophy of the liver is commoner than is generally thought; but as the condition in association with childbirth is usually the terminal phase of a toxæmia of pregnancy, it is possible that some deaths certified as due to eclampsia were due to this condition also.

Having this statistical information before us, the question arises as to what means are to be adopted to prevent or, if that is not possible, to diminish the number of deaths associated with pregnancy. Considerable attention has been paid to the subject recently and many different solutions have been offered. As they are of interest I mention a few of them. It has been said that reliance on political action in framing rules and regulations will improve the position and that the inauguration of a national maternity service, accompanied by the expenditure of much public money, will solve the problem. Then there are those who think that the solution rests with intensive instruction in the medical schools. There is also the view put forward that earlier treatment of complications, especially toxæmia, recognition of disproportion when it occurs, and early recognition of occipito-posterior positions will considerably reduce the death rate.

Where, then, does the solution lie? Is there one solitary factor responsible, or have many to be sought for? Are existing arrangements for the conduct of midwifery adequate, or should they be enlarged or altered?

At this point I wish to draw your attention to the so-called Rochdale experiment, which is probably familiar to you all and was reported in a recent issue of *The British Medical Journal* (February 16, 1935). The experiment consisted of an organized attempt to reduce the maternal mortality in the county borough of Rochdale, in Lancashire, one of the so-called "black" areas, where the maternal death rate was greater than 5 per 1,000 births, and in fact for the three years 1929 to 1931

was 8·9. This attempt was observed from 1932 to 1934, and during this three-year period the rate was reduced to 2·99. The authors state: "that the results were obtained by a change of spirit and method and without any alteration in the personnel or any substantial increase in public expenditure".

It is well to realize, in considering this result, that the death rate was exceptionally high before the attempt at reduction, and, as it is apparent from the study of some of the deaths detailed in this paper that all maternal deaths are not preventible, it would be interesting to know whether a similar procedure in any area where the death rate was not greater than 5 per 1,000 births would have had so pronounced an effect. I think it would produce a definite reduction in any area. Assuming this view to be correct, the question follows: Can the death rate be still further reduced in any other way? It would, I think, be possible to obtain this further reduction only by insuring that every confinement is conducted by a practitioner more highly trained and skilled than existing conditions can produce. Eardley Holland has pointed out in *The Lancet* of April 27, 1935, that practical training in midwifery must come after graduation. In order to provide this, training schools will have to be provided. There are a few of these in the British Empire, such as the Rotunda Hospital, Dublin, but they are so few that they hardly affect the position.

It appears, then, that a further reduction of the maternal death rate in favourable areas, that is, where the death rate is in the region of 3 per 1,000 births, is a different and more complicated task and one much more gradual of accomplishment than is the case with "black" areas.

In conclusion I wish to say that, in preference to outlining for you what I think is an ideal midwifery service, I have attempted in this paper to bring in aspects of the question which will promote discussion. I would also say that, pending political action of some sort, it is necessary for us as a profession to conduct our midwifery conscientiously and skilfully, so that we can face criticism when it comes, as it surely will, with a sense that our part of the midwifery service has been done, not only well, but to the best of our ability.

I should like to acknowledge with grateful appreciation the helpful assistance of the Registrar-General of Queensland in compiling the statistics.

MATERNAL MORTALITY: SOME PRACTICAL POINTS IN PREVENTION.[1]

By M. ELLIOT SMITH, M.B., Ch.M. (Sydney), L.M. (Dublin), F.R.C.S. (Edinburgh), F.R.A.C.S., M.C.O.G.,
Honorary In-Patient Obstetrician, Lady Bowen Hospital; Relieving Gynæcologist, Brisbane Hospital.

So many comprehensive orations have been given on the subject of maternal mortality that one is rather at a loss to produce something new and something which has not become hackneyed by constant repetition. One feels, too, that maternal welfare would be a better term to use for public expressions on the subject, as this would give to expectant mothers the psychological assistance of hope rather than the depressing and gloomy expectation of disaster.

I feel sure that this attitude of mind during pregnancy is of importance. More and more we find that patients fear pregnancy, and it is our duty to use every means at our disposal in teaching a more optimistic attitude to maternity.

I shall confine my remarks to two aspects of my subject only: (i) antenatal care, (ii) obstetric operations.

Since the late Dr. Ballantyne first pointed out the importance of antenatal care, its practice has become widespread. But the maternal mortality figures have not shown a corresponding decrease. Should we therefore condemn antenatal care as a means of reducing the mortality figures? Certainly not. For if we examine any number of deaths in childbirth, we are immediately struck with the fact that large numbers could have been prevented by early adequate treatment. Now just what does antenatal care include? To my mind it includes (I use the words of Dr. Jellett):

1. Giving the pregnant woman such information and instruction as will help her to live a normal and healthy life.
2. To discover the presence of any maternal abnormality or of any complication of pregnancy or labour.
3. To treat such abnormality or complication under the most favourable conditions and in the most satisfactory manner.

You may say that these three points are too obvious to be worth repetition, but the neglect of one or all of them so often leads on to disaster that I feel they cannot be too often repeated.

To day the medical profession as a whole is very alive to the value of antenatal care, but too often we lack the cooperation of the patients themselves and of their friends and relatives. The first problem then is to persuade the expectant mother to consult the doctor of her choice early in pregnancy and to follow out any instructions he may see fit to give her. It is essential to gain the patient's confidence and to instruct her to take no notice of any friend or relative who may have accumulated a fund of obstetrical curiosities specially designed to frighten the young mother.

I think public propaganda, if the welfare aspect were always emphasized, would soon overcome the frequent objections to antenatal examination and treatment.

Having enticed the expectant mother early in her pregnancy to the consulting room, we must make a careful investigation to discover any condition which may affect the health of the mother or child, or any maternal condition which may be made worse by pregnancy.

1. The general health must be investigated and a sane, sensible *régime* of moderate normal life must be impressed upon the patient.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on July 5, 1935.

2. The severe systemic diseases must be looked for—cardiac, renal and pulmonary.

3. The minor disorders of pregnancy or those minor disorders frequently associated with pregnancy are often present and warrant early treatment. I refer to constipation, indigestion, bacilluria, any of which may be caused or aggravated by septic foci in teeth or tonsils. Treatment of these minor disorders should be instituted and septic foci, if any, should be removed. The patient should always be sent to her dentist early in pregnancy and once again three months later to have the mouth put in perfect order. I have several times removed septic tonsils in early pregnancy and have seen no ill-effects. If there is anything in autogenous infection, then the removal of septic tonsils is an essential preventive measure.

4. Any gross deformity of pelvis or abnormality of pelvic organs should be detected at the first examination; this includes deformed pelvis, fortunately rare here in Australia, pelvic tumours, fibroids, ovarian cysts and retroversions of the uterus. Retroversions require replacement, if necessary under an anæsthetic, and the insertion of a pessary. Tumours must be treated on their merits. It may be best to leave them alone unless some complication supervenes.

5. In multiparous patients the past obstetric history is of great importance in deciding the possibility of an abnormal labour.

6. Having performed the foregoing, we have only two other duties during the antenatal period: (*a*) Periodic examination of the urine and taking of the blood pressure once a month for the first six months, then once a fortnight, and weekly in the last month. There is no need nowadays to stress this matter further, except that I do think the blood pressure reading is quite as important as the urine testing and may give us an early warning. (*b*) In the last four weeks to ascertain the presentation and position of the child and the presence or absence of disproportion. If the presentation be a breech or transverse, it can nearly always be corrected, and the same applies to occipito-posterior positions. A history of previous difficulty in *multiparæ* and non-fixation of the head in the last four weeks in *primiparæ* indicate need for hospital treatment.

Antenatal care and examination constitute a large field of preventive medicine, if performed thoroughly and practised wisely. Antenatal care enables us to divide cases into those which are normal and those in which some abnormality is anticipated. This division of cases must inevitably neglect accidental hæmorrhage and other pure accidents of pregnancy.

The first group, the normal cases, must be allowed to remain normal as far as is compatible with giving the patient the relief from pain which she desires.

For all cases in which difficult or operative delivery is to be expected, confinement in hospital should be insisted upon. None of us would dream of performing a major surgical operation except in a properly equipped operating theatre, and obstetric operations are to be regarded in the same light. If pregnant women are thus supervised and suitably treated, the severe toxæmias seldom develop.

In 1927, at Lady Bowen Hospital antenatal clinic, I saw 540 women at least twice during their pregnancy. Of these, 88 showed some degree of pregnancy kidney (not including pyelitis). In 23 the condition was severe enough to require in-patient treatment, that is, 4·26%. None of these patients developed eclampsia.

During the two years ended December 31, 1934, there were 13 deaths in the Lady Bowen Hospital. Of these, five were due to eclampsia, five were due to chronic nephritis, one was due to heart disease (mitral stenosis), one was due to influenzal pneumonia, one was due to hydronephrosis and pyelonephritis. There were no deaths from sepsis or from hæmorrhages.

Eight of these deaths were definitely due to associated diseases which had been made worse by pregnancy. With the exception of the patient with pneumonia, it is probable that all these patients would have lived with adequate supervision and early induction of labour.

In the five eclamptic cases death was directly attributable to pregnancy, but it is not unreasonable to assume that at least half of these patients would have lived if they had submitted themselves for prenatal supervision. I should like to say that they could all have been saved, but there is always an occasional fulminating case which gives no warning. Yet these cases are rare.

One feels that the interests of the State would have been served had the chronic nephritis cases been detected before pregnancy and had pregnancy been avoided.

The whole scheme of prevention of maternal mortality centres round the five major causes of death: (i) infection, (ii) toxæmias, (iii) hæmorrhage, (iv) dystocia, (v) trauma.

It has been clearly demonstrated that the death rate among women confined under the best hospital conditions is much lower than among those confined in other circumstances, and especially is this true of infection.

It is also established that by adequate prenatal supervision almost all the actual causes of mortality can be eliminated or removed by treatment before disaster threatens.

To sum up, deaths from toxæmias could be practically eliminated by antenatal care, deaths from dystocia and trauma decreased by early diagnosis and hospital treatment, and patients with *ante partum* hæmorrhage would also stand a better chance under hospital conditions.

The question of the prevention of infection is not a separate problem. It is intimately connected with the problems of dystocia and trauma and with the conduct in general of obstetric operations.

Obstetric operations should be just as safe as any other operation. There are two important

factors to consider: (i) A wise selection of the type of operation necessary in any particular case and judgement in deciding when to do it. (ii) The environment and circumstances in which the operation is performed.

If labour departs from the normal, the duty of the obstetrician is to aid Nature by restoring the normal mechanism, but it is not always necessary or advisable to do more than this. As an example I might quote the frequent problem of the occipito-posterior position. With the cervix short of full dilatation the head may, if necessary, be rotated to the anterior position (restoration of the normal mechanism) and the rest left to Nature. Forcible delivery following rotation will undoubtedly lead to trauma and aid the onset of infection.

In cases of *placenta prævia* the important object is to control the hæmorrhage, but not to deliver the child, as rapidly as possible. The softened cervix is easily torn in rapid delivery and infection frequently follows.

When a retained placenta is met with, expression under an anæsthetic is frequently successful, or the injection of glycerine into the uterus may enable one to avoid that most dangerous operation, manual removal of the placenta. In our obstetric section records of four deaths from sepsis, two followed this operation. The important third stage should not be hurried in the absence of hæmorrhage. Our object is to empty the uterus naturally and to leave it firm and well contracted.

In public hospitals all over the world the number of failed forceps cases admitted is high. From the number of these patients who deliver themselves naturally, it is obvious that the most suitable time for the application of forceps is not always chosen. And a very large number of the patients who have had forceps applied without success have a morbid puerperium and a few die. Is this just a part of the age of "hurry up" in which we live? And, if so, is the fault to be laid at the door of the medical practitioner or of the public?

It has been suggested that obstetric work should be taken away from the general practitioner and that a maternity service of midwives and obstetric specialists should be provided. This is unfair to the general practitioner. There are already too many inroads into his legitimate sphere of practice. I have heard it stated regretfully that there is no State medical organization to supervise children from the age of two years up to school age. I suppose when that is provided private practitioners will come into their own only when their patients reach adult age. If such a maternity service be provided, then our most valued and honoured institution, the family doctor, will indeed be a thing of the past.

The education of the medical practitioner has often been blamed for hurried midwifery. But to my mind the instruction received in our Australian medical schools is now perfectly adequate. The trouble more often is that the busy general practitioner lacks the time and opportunity to carry out the principles he has learnt.

The public still expects and, indeed, demands speedy delivery. It is time people were instructed that hurried midwifery is not the most desirable or the safest procedure; and it is time they were taught to allow the practitioner to exercise the deliberate patience that he knows is best for the mother.

But by force of circumstance the busy practitioner is often led to meet the demands of the public half way and to hurry on the delivery to allow him to attend other frequent and urgent demands for his services.

The remedy for this, to my mind, is the encouragement of group practice. I do not mean specialist group practice, but a group of three or more general practitioners working together in a district from a common consultation centre. Then, if one member of the group be detained all day by a tedious confinement, he need have no necessity to hurry off for other work, nor need he lose money by his inability to attend urgent calls.

Here in Queensland, with a State medical service on the horizon, such a suggestion might be given practical application. And this leads us to speak briefly of the economic factor. Obstetric work in industrial class practice is poorly paid for the amount of effort that has to be put into the work. If a consultant is required, the additional fee usually has to be subtracted from the attending doctor's fee, and so practitioners have to struggle single handed against the difficulties which arise.

It should be possible for practitioners, in their difficult obstetric cases, to send the patients into public hospitals and to continue to attend them there, if they wish, under the best conditions and with the assistance of specialist obstetricians.

I have said before that obstetric operations should be just as safe as any other surgical procedure. We would not contemplate opening an abdomen after a perfunctory preparation of the skin by a swab dipped in a weak solution of lysol. The female external genitals may be difficult to sterilize thoroughly, but by a free use of efficient antiseptics and by painting of the lower part of the abdomen, vulva and thighs with a suitable preparation, obstetric operations could be made safer for the mother. It is scarcely necessary to mention that gloves should be worn.

With a wise selection of cases and an early appreciation of abnormalities during labour the majority of these operations would be done in the best hospital environment or, failing that, in surroundings made as ideal as possible. Strict surgical cleanliness should be obtainable, an anæsthetist provided, and preparations made to meet all emergencies.

Patients for treatment by Cæsarean section need more careful selection. This operation is not an easy way out of all obstetric difficulties, but has certain very definite indications:

1. Many cases of disproportion due to small pelvis or pelvic tumours are best treated by this method.

2. Patients with eclampsia, in my experience, never require Cæsarean section.

3. *Placenta prævia* very rarely justifies Cæsarean section.

4. Abnormal presentations and positions never require Cæsarean section for the presentation alone. An associated small pelvis is a different matter.

5. Severe toxæmic hæmorrhages (*ante partum*) in some cases require Cæsarean section, but one must be prepared for hysterectomy.

This operation is a rapid and spectacular method of terminating pregnancy, but it carries with it considerable additional risk to the mother, even when performed under the very best conditions.

General practitioners should be encouraged to seek the aid of obstetricians in difficult cases. The public tends to regard the calling of a consultant in obstetrics as an admission of inefficiency on the part of the attending doctor. In actual fact it is generally the more efficient man who detects dangers before they arise and who obtains help for their prevention or treatment.

General surgeons are not the best consultants for obstetric cases. Their whole training leads them to look upon midwifery as a surgical problem, and the natural corollary is a Cæsarean section.

In this short paper I have attempted to present to you the major problems of prevention in obstetrics as it appears to me.

But I submit that the main object of this paper is not informative or instructive. If our minds are stirred to think along preventive lines or even to think deeply about the subject of maternal mortality, the object of these papers has been accomplished.

THE USE OF COMPOSITE FILTERS IN X RAY THERAPY.[1]

By C. E. EDDY, D.Sc., F.Inst.P.,
Research Physicist, University of Melbourne,

AND

JOHN O'SULLIVAN, M.D., B.S., D.M.R. and E.,
Honorary Radiologist, Saint Vincent's Hospital; Honorary Radiologist, Eye and Ear Hospital, Melbourne.

IN the treatment of deep-seated tumours by X radiation an increase in the penetrating power of the X ray beam results in: (*a*) a larger ratio of depth to surface dose, and (*b*) a more uniform dosage over a volume of deep-seated tissue. An increase in the penetrating power of the X ray beam can be produced by the use of higher excitation voltages and by increased filtration. The amount of filtration required to give a maximum percentage depth dose for the beam produced with any given voltage can be determined from physical measurements.[(1)] The manner in which the filtration is effected can have, however, important economic results.

The purpose of a filter is to absorb the useless long wave-length radiation with a minimum absorption of the useful shorter wave-lengths. The X ray energy is removed by the filter as a result of two independent processes: (*a*) a true or photo-electric absorption, (*b*) scattering. The longer wave-lengths are absorbed mainly through the photo-electric process, and the region of this absorption tends towards the shorter wave-lengths, with increase in the atomic number of the absorber. In the scattering process all the wave-lengths in the beam may be absorbed to approximately the same extent. For a minimum reduction in the intensity of short wave-lengths, therefore, it is necessary that the filter should absorb energy rather as a result of the photo-electric process than by scattering. Such a result can be achieved by a suitable choice of absorbing materials.

It is usual in diagnosis and in superficial therapy to employ filters of aluminium or of some other element of low atomic number. With voltages above 120 kilovolts it is customary to include as a filtering material an element of medium atomic number, such as copper, zinc or iron. The introduction of an element of higher atomic number instead of additional aluminium increases the proportion of energy lost by photo-electric absorption and decreases that lost by scattering, therefore permitting the passage of a greater amount of shorter wave-length radiation and giving a beam of greater penetrating power. It has been pointed out by Thoraeus[(2)] that for voltages above about 150 kilovolts the use of elements of even higher atomic number has important practical advantages. He suggested the use of a filter made up of a thickness of tin *plus* 0·25 millimetre of copper *plus* 1·0 millimetre of aluminium. He found experimentally that when the thickness of tin was adjusted so that the beam produced by the composite filter had the same half value layer (that is, was of the same quality) as that from a given copper filter, the tin-filtered beam had an appreciably greater intensity.

For voltages between 150 and 200 kilovolts constant potential Thoraeus advocated the use of a composite filter of 0·44 millimetre of tin *plus* 0·25 millimetre of copper *plus* 1·0 millimetre of aluminium. These two filters produced beams of the same quality (as measured by the half value layer), but that from the composite filter was 25% more intense. The use of such a filter therefore has considerable economic advantages. That it is equally satisfactory from the therapeutic standpoint can be inferred from the fact that such a method of filtration has been in use at the Radiumhemmet in Stockholm for some years.

We have recently constructed a Thoraeus composite filter including 0·44 millimetre of tin and

[1] Read at the Sixth Australian Cancer Conference, Canberra, May, 1935.

have compared the beam produced by this filter with that produced by one of 2·0 millimetres of copper and 1·0 millimetre of aluminium when used in conjunction with an X ray tube operated by a 200 kilovolt constant potential plant. A Hammer dosimeter was used to measure the dose delivered by each filter at the surface of a phantom of paraffin wax, and at a depth of 10·0 centimetres, the tube operating conditions remaining constant throughout. The results obtained are shown in the accompanying table. The average of six determinations of the time required for a dose of 5*r* is given, together with the mean departure of the mean. The concordance of these determinations is evidence of the steadiness of the X ray plant in operation and indicates that the difference observed for the two filters is not due to experimental error.

Comparison of Doses Delivered through Thoraeus and Copper Filters.

(Distance of surface of phantom from focal spot: 43 centimetres. Area of field: 48 square centimetres. Tube operated at 4 milliampères at 196 kilovolt constant potential.)

Observation.	Thoraeus Filter.	Copper Filter.
	Seconds.	Seconds.
Average time for 5*r* at surface	24·5±0·3	27·9±0·2
Average time for 5*r* at 10 centimetres depth	67·9±0·2	76·0±0·8
Time for 100*r* at surface	490	558
Percentage depth dose	36·1	36·7

It can be seen that the two beams produced an approximately equal percentage depth dose. To deliver any given surface dose, however, a time 14% longer was required when the copper filter was used.

It is therefore obvious that for the same number of working hours 14% more patients can be treated under the conditions given above by adopting the composite filter. This advantage is very important in busy hospitals with large waiting lists. Its advantage in private practice, due to the increased number of treatments obtainable from X ray tubes and valves, is no less important.

Similar increases in dosage as a result of the substitution of an appropriate composite filter for one of copper have been recently observed by several workers.[3][4] Some small discrepancies between the actual measured increases can be explained by differences in the value of the applied voltage, of the wave form of the high potential generator, in the type of tube used, and in focal skin distance and area of field for which the comparisons were carried out.

Thoraeus has shown that with increase in the penetrating power of the X ray beam, either through higher voltage or increased filtration, the superiority of a composite filter over a copper filter increases appreciably. From his results the thickness of tin required to obtain a composite filter equivalent in its quality-producing property to any given copper filter can be determined. From these figures it appears that a 3·0 millimetre copper filter can be replaced by one consisting of 0·6 millimetre of tin *plus* 0·25 millimetre of copper with an increase of nearly 30% in intensity.

Sheets of pure tin of any desired thickness can be readily obtained. In assembling the filter it should be pointed out that the tin component is placed nearest the X ray tube and the aluminium nearest the patient. The 0·25 millimetre of copper is sufficient to absorb those softer radiations (including the K radiations of tin) which pass through the tin filter, and the 1·0 millimetre of aluminium serves the same purpose for soft secondary radiation generated in the copper filter.

References.

(1) C. E. Eddy: "Recent Developments in the Production and Measurement of X-Rays", Report of the Fifth Australian Cancer Conference, 1934, page 42.
(2) R. Thoraeus: "Acta Radiologica", Supplement, Volume XV, 1932; "Tin Filters in Röntgen Therapy", *Ibidem*, Volume XV, 1934, page 225.
(3) Edith H. Quimby and L. D. Marinelli: "Influence of Filtration on Surface and Depth Intensities of 200 K.V. X-Rays", *Radiology*, Volume XXI, 1933, page 21.
(4) A. Mutscheller: "Composite X-Ray Filters", *Radiology*, Volume XXII, 1934, page 569.

Reports of Cases.

AN UNUSUAL INTRACRANIAL EMERGENCY.

By GILBERT PHILLIPS, M.Sc., M.B., B.S.,
Honorary Neurological Surgeon, Lewisham Hospital, Sydney.

OWING to the interest taken in recent years in intravenous methods of cerebral dehydration and the excellent results which have been produced by intravenous injections of hypertonic saline and glucose solutions in cases of acute cerebral trauma, it is of value to report the following case, in which an acute focal intracranial lesion, probably inflammatory in nature, by producing a high intracranial tension, both endangered the patient's life and in consequence of the disturbance of cerebral vascular supply did not allow the natural defence mechanisms to play their full part in attacking the inflammatory focus.

The patient, a medical student, aged twenty-seven years, was admitted to Lewisham Hospital on March 7 last. Dr. Wilfred Evans, who had been attending him, wrote that the patient had reported to him first on February 23, 1935, complaining of intense headache for a week and had stated that he had had similar headaches since Easter, 1934, and on one occasion it had been necessary to administer morphine. Dr. Evans thought that the intake of alcohol had precipitated the headache. On physical examination the patient's systolic blood pressure was 110 and his diastolic blood pressure was 80 millimetres of mercury, and Dr. Evans could find no abnormality in the heart or lungs. The urine contained no albumin or sugar. The patient had informed Dr. Evans that a Wassermann test had been made and that no reaction had occurred, and that his nasal sinuses had been examined by X rays with negative results. Careful examination of his nervous system had revealed no definite abnormality whatever, but Dr. Evans thought that the retinal veins appeared to be a little fuller than normal, and he had suggested that the patient should consult an eye specialist and have his fields of vision examined. A few days later Dr. James Flynn had told Dr. Evans that he could find nothing definitely pathological in the patient's fundi and that he was asking him to report again during the following week for further examination. It was then that Dr. Flynn first discovered a small hæmorrhage, but the disks were still normal. Two days later Dr. Flynn reported a definite papilloedema accompanied by increasing drowsiness.

On admission to Lewisham Hospital on March 7, 1935, the patient was conscious, well orientated and expressed

his apprehension of a possible craniotomy. He slept well that night and became drowsy the next day. When I first saw him at 4.30 p.m. on March 8, 1935, he was obviously passing into deep coma. He could not be roused either by forcible supraorbital pressure or by pin prick. Bilateral papillœdema (right three diopters, left four diopters) was present with retinal hæmorrhages in each fundus. Lumbar puncture revealed clear cerebro-spinal fluid at a pressure of 220 millimetres (over 100% increase). This pressure was slowly lowered to normal, and within ten minutes the patient was conscious and, except for some slurring of speech, was conversing normally with his mother. He answered questions intelligently and cooperated well during physical examination. His temperature was normal, but a blood count, made immediately, revealed a leucocytosis of 21,000, 93% of the cells being polymorphonuclear cells. Examination of the cerebro-spinal fluid disclosed no increase in globulin or cells, and no organisms were seen in smears made from a centrifuged deposit.

Within one hour of lumbar puncture the patient again became drowsy and then comatose. Fifty cubic centimetres of 50% glucose solution were injected intravenously and ten units of insulin subcutaneously, and again within a few minutes the patient was quite conscious.

This condition of progressive stupor relieved by intravenous dehydration continued for two days until March 10, 1935, and during this time the following intravenous injections were given, the indication for each one being impending coma, judged in each instance by increasing difficulty in rousing him by painful stimuli.

March 8, 1935—
- 5.30 p.m.—50 cubic centimetres of 50% glucose solution (with insulin).
- 8.30 p.m.—50 cubic centimetres of glucose solution.

March 9, 1935—
- 12.30 a.m.—75 cubic centimetres of 15% sodium chloride solution.
- 6 a.m.—75 cubic centimetres of 15% sodium chloride solution.
- 10 a.m.—50 cubic centimetres of 50% glucose solution.
- 10 p.m.—50 cubic centimetres of 50% glucose solution (with insulin).

March 10, 1935—
- 12 noon.—75 cubic centimetres of 15% sodium chloride solution.

After these injections it was found that unconsciousness could be controlled by concentrated magnesium sulphate enemata night and morning. In the intervals of consciousness succeeding each intravenous injection opportunity was taken of making full neurological examinations, and on March 9, 1935, right lower facial weakness was detected and the patient complained that he was having some difficulty with food, which tended to accumulate between the teeth and the right cheek. He also stated that he found it difficult getting his tongue in to remove the accumulated food. This weakness and disability passed off in the succeeding forty-eight hours.

Successive blood leucocyte counts were as follows: March 8, 1935: leucocytes, 21,000; polymorphonuclear cells, 93%. March 9, 1935: leucocytes, 21,500; polymorphonuclear cells, 90%. March 11, 1935: leucocytes, 14,000; polymorphonuclear cells, 80%. March 19, 1935: leucocytes, 14,400; polymorphonuclear cells, 76%. March 25, 1935: leucocytes, 11,600; polymorphonuclear cells, 84%.

On March 11, 1935, stereoscopic skiagrams showed no abnormality of the cranium and no signs of increased intracranial pressure of long standing. The paranasal sinuses were clear. On March 20, 1935, the visual acuity was $^6/_6$ in each eye and the visual fields were full. As a result of the subsiding papillœdema the blind spots were somewhat enlarged. Papillœdema had commenced to subside on March 12, 1935, and from that time the patient made a complete and uneventful recovery. At the present time (June 26, 1935) the disks are normal, except for the absence of physiological cupping, the retinal hæmorrhages have absorbed, there is no sign of right lower facial weakness and no other abnormal neurological signs can be detected. The patient feels extremely well, has put on over a stone in weight, and has resumed his medical studies.

Comment.

The early finding of a cerebro-spinal fluid pressure of 220 millimetres indicated that coma was secondary to some intracranial catastrophe. Intracranial tumour appeared to be quite unlikely from the nature of the history and from the absence of prodromal neurological signs in a patient passing so dramatically into coma. A hint as to the nature of the lesion was given by the leucocytosis of 21,000 with a neutrophilia of 93%. That the lesion was a local and not a general one was shown by the normal cerebro-spinal fluid. Its localization to the left temporal lobe was indicated by the right lower facial weakness, and it is well recognized by those who have had experience with intracranial neoplasms that lesions in this situation are particularly prone to cause disturbances of the cerebro-spinal fluid circulation. I shall not be so unwise on such slender evidence to insist on the diagnosis which I believe to be the correct one, but I do feel sure that the lesion was a focal inflammatory one. A primary septic focus was found in the jaw, an X ray report of which read as follows: "There is a very extensive apical sepsis and absorption of the lower left first molar." The patient admitted afterwards that he had had a great deal of trouble with this tooth over a long period, and for some time had had a sinus pouring pus into the mouth from the apex of the tooth. This sinus had become blocked intermittently. There can be no doubt that intravenous dehydration was of absolute value in saving this patient's life, since Cheyne-Stokes respiration appeared on two occasions when the intravenous injections were somewhat delayed. If, as I believe, the lesion was an inflammatory focus, the presumption is strong that the restoration of normal intracranial vascular and cerebro-spinal fluid circulations by brain shrinkage enabled the normal powers of resistance to attack and defeat the lesion at an early stage of its development.

Acknowledgements.

I am indebted to Dr. Wilfred Evans and Dr. James Flynn for the early notes of this case.

QUININE AMAUROSIS.

By GEORGE SIMPSON, M.B., B.S. (Melbourne), M.R.C.P. (London), Dip.G.O. (Melbourne),
Honorary Assistant Surgeon, Women's Hospital, Melbourne,

AND

ARCHIE S. ANDERSON, M.B., B.S. (Melbourne), D.O.M.S. (London), F.R.A.C.S.,
Honorary Ophthalmic Surgeon, Victorian Eye and Ear Hospital, Melbourne.

THE following case of quinine amaurosis appears to be worth recording on account of the comparative rarity of the condition, the completeness of the recovery, and the absence of any abnormality in the fundus picture, even when the amblyopia was very marked.

The patient, a married woman, aged thirty-six years, had previously been confined twice by one of us (G.S.). On each occasion labour had been very difficult on account of post-maturity and a large baby. Both times she had induction by quinine and pituitrin without untoward effect.

At 9 p.m. on May 10, 1935, she took a dessertspoonful (approximately 50 grains or 3·5 grammes) of quinine sulphate, with the idea of terminating an unwanted pregnancy. She woke next morning completely blind, but with no tinnitus or other sign of quinine poisoning.

At 10 a.m. she was unable to recognize light from dark, and her pupils were widely dilated and fixed. The fundi were not examined.

At 12.30 p.m. the pupils were still widely dilated and fixed, but her vision had improved to the extent of being able to distinguish the forms of those around her. At times she could even see their faces as white blotches, but this was only in momentary and fleeting periods. Her fundi were examined by one of us (A.S.A.) and found to be entirely normal in appearance—no retinal œdema and no constriction of arteries were present.

She was given a mixture containing digitalis and strychnine. Her vision gradually and steadily improved and by the next morning it appeared to be normal and the pupils were normal in size and reactions. Eighteen days later a detailed ocular examination was made. The pupils were found to be equal and of normal reactions; the fundi presented no abnormal appearance; the vision was $^6/_4$ in each eye and Jaegar 1; the fields, tested with a one millimetre white object at 33 centimetres, were full in extent.

Comment.

The dose, though large, is by no means without its equal in the literature.

Gainsborough[1] reports a case in which the patient took four grammes (60 grains) in one dose, became completely blind in three hours, but ten hours later was able to see normally.

Evans's[2] patient showed changes in both fundi two months after swallowing 7·5 grammes (100 grains).

Ballock and Lagrange[3] record a case in which the taking of three grammes produced total blindness; the fields were permanently constricted to the 5° circle.

Amat Marin's[4] patient took one gramme every day for five days. Recovery was complete for a time, but three months later the arteries showed some constriction.

But easily the biggest dose found in the literature was that recorded by Musial;[5] in this instance twenty grammes were taken at a single dose. The vision finally was $^6/_6$, but there was a field loss to the 20° circle.

Though the text books suggest that arterial constriction is a constant finding in these cases, quite a number of records mention the entire absence of any such appearance. Duggan and Navati[6] saw one patient in whom there was retinal œdema but no change in the vessels.

Scardapane[7] notes three cases in which the fundus details were normal, though in each there was contraction of the field. The same writer[8] collected twenty cases from the literature, in all of which the fundi appeared to be normal. This leads him to champion the idea that quinine acts by direct toxic action on the visual nerve elements rather than by way of anæmia secondary to arterial constriction. In this contention he receives the solid support of Traquair,[9] who marshals several convincing clinical facts to support his belief.

References.

[1] H. and R. Gainsborough: "A Case of Quinine Amblyopia", *British Journal of Ophthalmology*, Volume VI, 1922, page 259 (abstracted in "Ophthalmic Year Book", 1923, page 232).

[2] J. N. Evans: "Quinin Amblyopia", *American Journal of Ophthalmology*, Volume VI, April, 1923, page 271.

[3] Ballock and Lagrange: "Visual Disturbance in Quinin Poisoning", *American Journal of Ophthalmology*, Volume VI, October, 1923, page 852 (abstracted in "Ophthalmic Year Book", 1924, page 251).

[4] Amat M. Marin: "Quinine Amaurosis", *International Survey of Ophthalmology*, Volume VI, 1923, page 22 (abstracted in "Ophthalmic Year Book", 1924, page 251).

[5] A. Musial: "Temporary Blindness after Quinine Intoxication", *Polska gazeta lekarska*, Volume II, 1923, page 525 (abstracted in "Ophthalmic Year Book", 1924, page 251).

[6] Duggan and Navati: "Quinine Amblyopia with an Unusual Fundus Picture", *American Archives of Ophthalmology*, Volume IV, August, 1930, page 265.

[7] F. Scardapane: "Blindness and Amblyopia from Quinine", *Saggi di oftalmologia*, Volume V, 1930, page 62 (abstracted in *American Archives of Ophthalmology*, Volume IV, October, 1930, page 575).

[8] F. Scardapane: "Experimental Study of Quinine Amblyopia", *Saggi di oftalmologia*, Volume V, 1930, page 240 (abstracted in *American Archives of Ophthalmology*, Volume V, January, 1931, page 135).

[9] H. M. Traquair: "Toxic Amblyopia including Retrobulbar Neuritis", *Transactions of the Ophthalmological Society of the United Kingdom*, Volume L, 1930, page 384.

EPIDEMIC CONJUNCTIVITIS.

By K. S. JONES, M.B., B.S. (Sydney),
Portland, New South Wales.

AN epidemic conjunctivitis has been evident in Portland since March, 1935, and is still in evidence at the present time. During this period there have been over one hundred cases in school children and at least as many cases again in non-school patients. There has not been a high incidence in the surrounding district, but in the town itself the disease has proved extremely contagious and often a whole family has become infected. People of all ages and of both sexes suffered impartially.

The usual clinical history was as follows. The patient came to the surgery complaining of a gritty feeling in one eye, followed by a purulent discharge and inflamed appearance of the eye the next day. On examination the eyelid presented slight œdema, the conjunctiva was thickened and inflamed, and some dried secretion was present at the inner canthus. On everting the eyelid small flakes of fibrin were usually found in the conjunctival sac. Untreated, the condition sometimes pursued a course of some weeks with mild irritant symptoms, but in the majority of cases the discharge greatly increased and the pain became severe enough to drive the patient to seek medical attention. In approximately 70% of cases the second eye was infected within a few days. With treatment the disease lasted two weeks on the average.

The following routine treatment was instituted: Instillations of a 20% solution of argyrol two to four hourly, according to the severity of the condition; an eye-bath of a 3% boric acid solution twice daily, and in the severe cases a light shield for the eye; local heat proved effective in combating any pain. This treatment was successful in the majority of cases, but some 5% of patients still had signs and symptoms after two weeks. In these cases the instillations were changed, a solution of zinc sulphate, 1 in 500, being used; this seemed to clear up any remaining infection.

A number of pus smears were examined by the Jensen modification of the Gram method of staining, and all of these showed the presence of Gram-positive diplococci resembling pneumococci. There were no organisms resembling the Koch-Weeks bacillus. The conclusion was drawn that the epidemic was probably due to an undetermined strain of pneumococcus of mild virulence, similar to other pneumococcal epidemics which are occasionally reported.

There may possibly be a local immunity conferred on the patient, since I have observed no recurrence of the disease in any patient clinically regarded as cured.

Reviews.

RESPIRATION.

"RESPIRATION", by J. S. Haldane and J. B. Priestley, is a thoroughly revised edition of a monograph by the senior author first published in 1922, which represents in an expanded form the Silliman Lectures delivered by him at Yale University in 1916.[1] Both the authors of the present

[1] "Respiration", by J. S. Haldane, C.H., M.D., F.R.S., and J. G. Priestley, M.C., D.M., M.A.; New Edition; 1935. Oxford: The Clarendon Press. Medium 8vo., pp. 493. Price: 30s. net.

volume have been prominently associated with experimental investigations on the subject. Of the first edition Haldane wrote: "The book sums up the results of more than twenty years of my own experimental work, thought, reading and investigation." Thirteen years have elapsed since this was written, so that he now surveys the field from the vantage point of more than half a lifetime. The book has been almost entirely rewritten, but headings of chapters are the same as in the first edition. Their order has been somewhat changed for the sake of greater continuity, and each chapter has been considerably lengthened by the inclusion of important work published during the past thirteen years. It is disappointing to find that much of the recent American work is omitted and that the description of modern views on oxygen secretion is incomplete. It is a pity, too, that the authors have not troubled to analyse some of the figures they quote. The table on page 148, for instance, will hardly stand critical analysis.

Haldane's point of view is still essentially different from that of most workers in this field. In the preface to the first edition he had written: "The mechanistic theory of life is now outworn and must soon take its place in history as a passing phase in the development of biology . . . I have tried to paint a picture of the body as alive." In the preface to the present edition, and, indeed, throughout the book, he emphasizes the necessity for this method of approach: "In rewriting this book we have endeavoured to represent more clearly and sharply than before that the physiology of respiration deals with phenomena which are specifically those of life."

"At present it is usual", he writes, "to treat the physiology of respiration as being nothing more than a description and analysis of all the physical and chemical processes by which oxygen is supplied to the living body and carbon dioxide removed from it. But when we survey such a description it is quite evident that something is missing from it. Not only is oxygen supplied and carbon dioxide removed, but these processes, as will be made evident in the course of the book, are co-ordinated at every stage among themselves and with other physiological activities in a manner which is characteristic for each organism. Moreover, the structures involved in the process are maintained in a characteristic co-ordinated form during adult life, and have developed into this characteristic form from a germ in which no such form can be distinguished. A mere physical and chemical description furnishes no account of this co-ordination, maintenance and development." Haldane claims that this point of view is not the vitalism of the older physiologists, but one cannot help feeling that it is still vitalism which has undergone, as it were, a considerable shift to the left, owing to the fact that our knowledge of the physics and chemistry of respiration is far greater than that of the older physiologists. There is still much that we cannot explain, and for this unexplained residue Haldane evokes what he calls "the specific conception of life", while physiologists of the mechanistic school assume that we shall in time be able to interpret all the phenomena of breathing in terms of physical science. To Haldane this is an unjustifiable philosophical assumption.

This point of view may seem to some to be a compromise; for how, in the present state of our knowledge, can we set a limit to what may be explained by physics and chemistry? It has the advantage, however, of being imaginative, elastic and, on the whole, humble, as compared with the rigid, literal and frequently limited outlook of the mechanistic biologist. Haldane considers that the endeavour to represent the facts of physiology as if they would fit into the general scheme of a mechanistic biology has led to the present estrangement between physiology and medicine. "It is only when we realize how real the co-ordination and maintenance are that we can apply physiology effectively to the diagnosis, treatment and prevention of disease." The great practical value of his scientific work has given him the right to make this assertion.

To the physiologist the earlier chapters, dealing with the physiology of respiration, constitute the most important part of the book, but the general medical practitioner will be more interested in the series of special chapters dealing with the application of the general conclusions to clinical medicine and various forms of industry.

Chapter VIII, on the causes of anoxæmia, is of great interest, dealing as it does with such widely divergent conditions as the shallow breathing of war neurasthenia, the periodic breathing of toxic states, the deficient oxygen pressure of high altitudes, the anoxæmia of over-exertion, and the nature of carbon monoxide poisoning. The authors stress the extreme importance of realizing the dangers of anoxæmia.

The subject of anoxæmia is pursued further in the chapter on "Effects of Low Atmospheric Pressures", such as are met with on mountains or high plateaux and in ascents by balloons or aeroplanes. Of special interest is an account of what is known of respiratory conditions in the stratosphere. In accordance with the philosophy of this book, the phenomena of acclimatization to low pressures are interpreted as a tendency of the living body to maintain its normal standards. The authors consider that acclimatization is due in part to increased power of oxygen secretion, as well as increased hæmoglobin percentage, and diminution of the available alkali in the blood and tissues so as to permit of increased breathing without the development of alkalosis. The extremely controversial matter of oxygen secretion in the lungs has a chapter to itself. It is a lengthy and somewhat involved chapter. One is fascinated by the detailed and beautifully illustrated account of oxygen secretion in the swim bladder of fishes, but one remains unconvinced that oxygen secretion has been proved to occur in the lungs. It is after all a far cry from the air bubbles of Arcella to the alveoli of the human lung—especially is it a far cry for one who asserts, as Haldane does in his preface, that human experiments are all-important and that "their rightful place has been usurped too long by experiments on fragments of frogs and other animals". Perhaps the chapter is unconvincing because the matter is as yet insufficiently investigated. In deference to a great physiologist it should be remarked that such analogies do not occur often in this book. Most of the investigations specially referred to have been carried out on man, many of them on Haldane himself, to his no small danger and discomfort.

The chapter on effects of high atmospheric pressures is concerned with conditions present in diving, tunnelling and submarine escape apparatus. The prevention and treatment of air embolism are fully discussed. The concluding chapter on air of abnormal composition is also of practical importance, as it deals with such subjects as ventilation, gas poisoning and the air of mines and tunnels. There is probably no greater living authority on this latter subject than Haldane, who since 1894 has been actively concerned in the improvement of conditions in coal mines.

It is a pleasure to comment on the excellence of the production and printing of the book. The text is freely illustrated by tables and diagrams and by several photographic plates, which are apposite and well reproduced. The chapters are rather long—there are only thirteen of them in a book of 456 pages—and, with the exception of unobtrusive variations in the page titles, there are none of the sub-headings, summaries or variations in print by which those inveterate standardizers, the Americans, are wont to enliven their work and save the time and thought of their readers. It is perhaps more dignified to rely, as the authors of the present volume have done, on large pages, excellent type, and a style which is for the most part lucid and emphatic, and which becomes involved only when the problems discussed are of such complexity that adequate discussion is incompatible with brevity and simplicity.

The Medical Journal of Australia

SATURDAY, AUGUST 31, 1935.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

TOXÆMIA OF PREGNANCY AND CARDIO-VASCULAR DISEASE.

REFERENCES have been made in these pages from time to time to some of the unsolved problems of hypertensive arterial disease, in particular the factors concerned in its causation. If the demonstration of the presence of pressor substances in the blood had been confirmed, there might have been some hope of a chemical solution of this problem of ætiology, but later workers have completely failed to find evidence of such substances. It would seem that investigation of cases of toxic gestation might be more likely to yield results in such an inquiry, since the material is drawn from a more restricted and definite field. Anselmino and Hoffman in 1931 reported that they had found evidence of the presence of a blood pressure raising and antidiuretic substance in the blood of patients suffering from eclampsia or preeclamptic toxæmia.

David Hurwitz and Lewis T. Bullock have just published work on the same subject, but their findings are in complete disagreement with those of the earlier observers.[1] They used ultra-filtrates from the blood of eleven eclamptic and preeclamptic patients and tested these for the presence of alleged pressor and antidiuretic bodies by injecting rabbits with the material and observing the effect on the blood pressure and on the diuresis noted normally after the introduction of water into the animal's stomach. It was found, incidentally, by control experiments that over half the available rabbits had to be discarded, since they did not show consistent diuresis curves. With these precautions the results were entirely negative. These observations fall in with those of F. B. Byron and C. Wilson, who last year investigated the same question.[2] Byron and Wilson pointed out that in addition to the reports of Anselmino and Hoffman referred to above, Küstner had demonstrated the effect of eclamptic serum on the melanophores of the frog's skin, and Cushing had claimed that an invasion of the basophile cells of the pituitary occurred in some hypertensive states, such as eclampsia. Therefore it was important to ascertain if the antidiuretic hormone of the pituitary could really be found in the blood of eclamptics. They rejected the rabbit as an experimental medium in favour of the rat, and used a method that will detect 0·001 international units per 100 grammes of rat. Titration with standard pituitary extract proved the reliability of this method, but even with so sensitive an indicator no trace of the antidiuretic hormone could be detected in the plasma of ten patients with preeclamptic toxæmia and three with eclampsia and œdema.

Although these researches have not led to any positive result, the association of eclampsia with hypertension is recognized as being very significant, since permanent cardio-vascular disease not infrequently follows eclampsia. It is known that sufferers from toxæmia of pregnancy who do not make perfect recoveries usually show evidence of the hypertensive syndrome; the aftermath is, in fact, far more often cardio-vascular than renal. This has been emphasized by W. W. Herrick and

[1] *The American Journal of the Medical Sciences*, May, 1935.
[2] *The Quarterly Journal of Medicine*, July, 1934.

A. J. B. Tillman in an article dealing with the results of a long follow-up investigation of a large series of such patients.[1] Nine hundred and thirty patients have been observed during a period of fifteen years, and, of these, 594 patients have been reexamined at least twice a year by physicians rather than by the obstetric members of the hospital staff. The average period of the follow-up was five years. It was found that the mortality in this group of 594 was 15·7%, which is a figure nearly seven times as great as the average death rate for women of child-bearing age. Of the 90 deaths, 72, that is no less than 80%, were due to cardio-vascular causes. The authors consider that the clinical evidence and the pathological findings, autopsy and otherwise, suggest a continuous chain of events in cases of toxæmia that lead on directly to cardio-vascular sequelæ. More than half the survivors of these toxic states revealed signs of either glomerulonephritis or hypertensive cardio-vascular disease within three years; of these two end states a latent or manifest primary nephritis was much the rarer, a point that may well engage the attention of the practitioner engaged in obstetric work, for his preoccupation with the urinary findings in the pre-natal period may tend to obscure the importance of hypertension after the confinement. If it is generally true that in over 50% of cases of eclampsia and preeclampsia the pathological changes are not transitory but lead to vascular or nephritic disease, the position is so serious that sufficient stress cannot be laid on the vital importance of adequate pre-natal care.

Current Comment.

POLYCYTHÆMIA VERA.

A CONDITION of *polycythæmia rubra* has been observed in cigarette smokers who suffer from chronic carbon monoxide poisoning. It also occurs in residents at high altitudes, where the oxygen tension of the atmosphere is diminished, necessitating an increase in the oxygen capacity of the blood. After arrival at high altitudes there is rapid proliferation of the red blood corpuscles. It has been estimated that in Bolivia, at an altitude of 15,400 feet, the red cell count both for Indians and whites reaches eight to nine millions per cubic millimetre. Animals are said to exhibit a similar increase. In congenital malformations of the heart the red cells are often increased, the body endeavouring to compensate for its oxygen deficiencies by increasing the number of oxygen-carrying corpuscles. In cyanosis associated with lung diseases a similar increase of red cells may be observed. The occurrence of polycythæmia in chronic bronchitis is considered a very unfavourable omen, often the precursor of death. A red cell increase may be seen in emphysema; but breathlessness is a more typical feature of emphysema than is cyanosis. These are all cases of true polycythæmia, the absolute number of corpuscles being increased owing to excessive erythroblastic activity of the bone marrow. Such examples, however, are secondary, compensatory or symptomatic. A red cell increase has also been noted in hypopituitarism and in non-bleeding duodenal (but rarely or never in gastric) ulcers. In the last mentioned cases it has been suggested that chronic pancreatic inflammation is in some vague way responsible. A relative polycythæmia may arise from concentration of the blood in conditions of severe diarrhœa or protracted vomiting.

Polycythæmia vera, known also as erythræmia, splenomegalic or myelopathic polycythæmia, or *erythrocytosis megalosplenica*, is a primary disease due to some disorder of the marrow of the long bones, but the ætiological factor thus acting is unknown. Males are more often affected than females and the usual age is between thirty and sixty. Polycythæmia is sometimes seen in syphilitic splenomegaly, but it is doubtful whether true erythræmia is ever a cause of chronic splenomegaly in childhood. Amongst the various manifestations is an increase in the viscosity of the blood with prolonged coagulation time. Hæmorrhages are frequently seen. Vascular thrombosis may be present with hemiplegia when cerebral vessels are involved. There is increased fragility of the red corpuscles. The total volume of the circulating blood is increased. The number of red cells averages about nine millions per cubic millimetre, but may reach thirteen millions. The hæmoglobin percentage may be 124, but the colour index may be as low as 0·7. The blood platelets are moderately increased and a leucocytosis is frequent, with a slight increase in the polymorphonuclear cells. A leuchæmic blood picture may supervene, with progressive enlargement of the spleen. In one recorded case the condition passed into one resembling pernicious anæmia. Ophthalmoscopic examination may reveal dilatation of the retinal veins, with deepening of their colour so that they appear as black streaks. The normal colour of the eye-grounds may be replaced by a cyanotic hue. The arteries display no characteristic change. There may or may not be choked disk or retinal hæmorrhages. The spleen is enlarged, but may ultimately shrink and become no

[1] *Archives of Internal Medicine*, April, 1935.

longer palpable. Hepatic enlargement is often present.

One of the outstanding features of the disease is a cherry-red cyanosis of the face, lips, buccal mucosa and hands, accompanied by visible venules. Sometimes arteriosclerosis with high blood pressure is associated with the disorder, but not invariably, and there may be no enlargement of the heart or any evidence of cardiac disturbance. The manifestations of the disease are so protean that errors of diagnosis are common and differential diagnosis may be difficult.

In view of the difficulty in diagnosis, attention should be drawn to a report of nine cases by L. J. Adams, in which he makes special reference to nervous manifestations.[1] He points out that Vaquez's original patient in 1892 had tinnitus and paroxysms of dizziness. In 1903 Osler mentioned nervous symptoms in his own cases and in those recorded in the literature. Of Adams's patients, five were males and four were females. One was twenty-five years of age and the oldest sixty-one; excluding the one of twenty-five, the average age was fifty at the onset of symptoms. The patients were under observation for periods ranging from six months to eleven years. The most characteristic symptoms were headache, dizziness and paræsthesiæ, with numbness in the fingers or toes, pains in the legs and disturbance of vision. Most of the patients sought advice for symptoms referable to the nervous system. The condition of one patient was incorrectly diagnosed as traumatic epilepsy, a previous head injury having confused the issue. This patient was subsequently subjected to operation elsewhere with the same diagnosis, but nothing abnormal was found. In several cases many other diseases have been wrongly diagnosed, such as arsenical neuritis, Banti's and Hodgkin's diseases, cirrhosis of the liver, gall-stones, chronic nephritis, and even epigastric hernia and intestinal obstruction. Pain and other abdominal manifestations have led to fruitless surgical intervention. A remarkable case, in which recurring hydrothorax, ascites and jaundice were present, resembled one reported by F. P. Weber, in which jaundice and hepatic cirrhosis were followed by fatal hæmatemesis. Hepatic enlargement occurred in most of Adams's cases, and splenomegaly in all. The heart was not enlarged in any. Three patients had hypertension. Abnormalities were seen in the ocular fundi of the four examined. Hæmorrhages, especially post-operative, occurred in all but one, and included hæmatemesis and hæmaturia.

[1] *The Canadian Medical Association Journal*, February, 1935.

BACTERIA AND GASTRIC ULCER.

ONE of the most difficult problems that await full solution in connexion with the pathology of gastric ulcer is the explanation of its tendency to chronicity. In fact all ulcerations in the alimentary tract tend both to recur and to become chronic. In the stomach and duodenum there is a factor which is unique and peculiar to these sites: the presence of hydrochloric acid. Whether this substance or the digestive ferments are responsible for the long continuation of once established ulcers has been debated. Some acute ulcers are known to heal rapidly, and the finding of multiple scars on the duodenal mucous membrane also shows that healing may sometimes proceed without interruption or delay. But the experimental method is necessary in order to investigate the problem from all angles, even allowing for the fallacy inherent in the application of the results to the human subject.

Samuel Morrison and Maurice Feldman have carried out some work on dogs which adds somewhat to what is known about gastric ulcerations.[1] They remark that it is agreed that acute ulcers may easily be produced without major operative procedures, and that these heal readily. What they set out to establish was the part played by bacterial infection in the maintenance of ulceration. To this end they produced acute ulcers by injecting the stomach wall with 1% hydrochloric acid and, when these ulcers were established, introduced bacteria with the food. It was thought that the food would dilute and neutralize the gastric contents and also that the bacteria would thus remain for longer periods in the stomach and be protected more or less from the action of the acid. In another group of animals bacteria were introduced alone, and in yet another group large amounts of bicarbonate of soda were given at the same time in order to neutralize the high acidity of the dog's stomach. The bacteria employed were *Staphylococcus aureus, Bacillus coli* and *Streptococcus viridans*. In some animals gastrostomy was carried out so that the incidence and progress of ulcers could be directly observed, and streptococci were inoculated directly into the ulcer walls. Even in the last-named cases healing occurred promptly, and this experience was reproduced in all the other series also; that is, in the dog chronic gastric ulceration could not be produced by bacteria alone or by bacteria acting on ulcers caused by hydrochloric acid. Of course, too much significance cannot be placed on such experiments—they are necessarily artificial—but the authors appear to be justified when they state that another factor must play an important part in the production of chronic gastric ulcer. The recognition of chronic gastritis in association with ulcer will come to the mind in this connexion, but it must be admitted that in these experiments the massive injection employed caused no definite lasting inflammation of the stomach. In any case Morrison and Feldman have done useful work in drawing attention to what seems to be the truth, that all the factors at work in causing the clinical disease of chronic gastric ulcer in man are not yet known.

[1] *The American Journal of the Medical Sciences*, May, 1935.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

The Oculo-Cardiac Reflex.

J. H. BAILEY (*American Journal of Ophthalmology*, January, 1935) reports the case of a man of thirty-one years, whose left eye was enucleated after injury and who exhibited the oculo-cardiac reflex. From the cut optic nerve free bleeding occurred, which was not controlled by pressure or heat. Accordingly the author inserted a continuous suture in the edges of the conjunctiva. The sutured conjunctiva, with the enclosed blood, protruded beyond the interpalpebral fissure. The patient soon complained of feeling faint, became pale and perspired freely and was nauseated and felt precordial distress. The pulse rate was 65, soon dropping to 60, then 55, and gradually till it reached 30 per minute. It was full and strong, though it would frequently lose a beat. The author's alarm was set at rest by suddenly remembering the oculo-cardiac reflex. The essential feature of this reflex (called sometimes the Dagnini-Ochsner reflex) is the retardation of the heart beat when adequate pressure is exerted upon the contents of the orbit. It varies in different individuals. In vagotonia the response is more intense; in sympatheticotonia the pulse may even be accelerated. Orbital pressure affects the branches of the first division of the fifth nerve; the stimulus travels to the Gasserian ganglion and thence to the pons and vagus centre.

Acute Dacryadenitis.

F. B. FRALICK (*American Journal of Ophthalmology*, January, 1935) describes the rather uncommon condition of acute inflammation of the lachrymal gland. The commonest cause is mumps, when the dacryadenitis is bilateral and does not suppurate. Other causes are gonorrhœa, syphilis, tuberculosis, exanthematous infections and generally any septic focus. The inflammation may resolve or proceed to suppuration. It begins with pain in the lachrymal region, swelling and œdema of the outer angle and drooping of the upper lid. There is lachrymation and then later purulent discharge. The patient may complain of diplopia and the eye may be turned inwards and downwards, with limitation of abduction. The preauricular gland may be enlarged. Iced compresses are useful at first, then heat.

Glaucoma Capsulare.

T. M. SHAPIRA (*American Journal of Ophthalmology*, January, 1935) reports three cases illustrating Vogt's *glaucoma capsulare*. A degenerative process takes place in the anterior lamella of the lens capsule. Movements of the iris rub off this material into the aqueous, whence it reaches and blocks up the angle, causing glaucoma. Slit-lamp examination reveals exfoliated material; there is a frost-like appearance of the pupillary border of the iris. Opacity of the lens and increased tension are present. Trephine operations are not so successful as in the usual cases of glaucoma.

Lens Extraction in Myopia.

A. ELSCHNIG (*American Journal of Ophthalmology*, December, 1934), though issuing a warning that myopic eyes have a predisposition to iridocyclitis, has evolved a technique for the extraction of the lens in myopia which he considers safe. It is noted that the emmetropic eye after lens extraction required a + 11 diopter for distance, yet an eye must be 20 diopters myopic to be emmetropic with the lens removed. This is a guide for the cases suitable for operation. The author does not operate on patients below ten years of age, and in patients up to twenty-five he performs discission; after twenty-five he prefers to extract in the capsule. The discission is made in the lower half of the dilated pupil so that in case the lens swells greatly the upper half of the anterior chamber will remain deep enough to permit a keratome incision. This is done after three or four days, the remaining capsule is lacerated and the lens is evacuated by suction. Eserine is then instilled. The remaining lens matter should be absorbed in two weeks. It may be necessary to open the chamber again and extract as much as possible. The author does not consider that the operation makes detachment of the retina more likely to occur.

Copper Ionization in Lachrymal Stenosis.

H. L. HILGARTNER, SENIOR AND JUNIOR (*American Journal of Ophthalmology*, January, 1935), report the case of a man of sixty-eight with intractable stenosis of the nasal duct. A copper wire the same size as a number 3 Theobald probe was passed into the nose and attached to the negative pole of a four milliampère current. This was continued for twenty minutes. Though the reaction was severe, a cure was effected.

Neuro-Retinitis from Ergot Poisoning.

D. KRAVITZ (*Archives of Ophthalmology*, February, 1935) relates the case of a woman who took ergot and suffered from loss of vision. The patient, twenty-two years of age, having missed a period, was given by a druggist pills containing one grain of ergot. She took two pills four times a day. On the fifth day she had pains in the head, and the next day her sight was blurred. The vision in the right eye was $^{20}/_{200}$; the vision in the left eye was $^{20}/_{30}$. The disks were pale salmon colour and elevated two diopters. The margins were indistinct and the retina surrounding them was œdematous. The fields were contracted, the blind spots enlarged and absolute central scotomata were present. For some weeks the ingestion of ergot was not revealed and an unsuccessful search was made for the ætiological factor.

Sutures for Lid Control in Cataract Operations.

W. D. HORNER (*American Journal of Ophthalmology*, January, 1935) considers that the perfect model for an eye speculum has yet to be designed. Not one can be said to eliminate pressure on the globe completely. The use of lid retractors requires a trained assistant. The author now uses a lid suture for upper and lower lids. At first a single suture was placed in the upper lid with a bite of six millimetres of skin in the centre; but it was found that better retraction was made with two sutures each with a bite of eight millimetres and starting three millimetres from the middle line. The four strands of silk were caught evenly in a straight catch forceps.

Eye Socket Reconstruction in Thyreoid Disease.

G. B. O'CONNOR AND G. W. PIERCE (*American Journal of Ophthalmology*, January, 1935) relate the case of a man of sixty who was unable to retain a glass eye after enucleation following injury. He gave the history of two operations for thyreoid disease. The mucosa of the socket was inflamed and œdematous and protruded more than normal because of a soft bulky mass behind it. This mass was removed and proved to be hypertrophied extrinsic eye muscle filling about three-fourths of the orbital cavity. The authors refer to the important work of H. C. Naffziger on the pathological changes in the extraocular eye muscles associated with thyreoid disease.

Nystagmus in Infants.

P. J. MOORAD (*Archives of Ophthalmology*, February, 1935) finds that monocular nystagmus in infants without the usually associated pathological states is uncommon. The horizontal type is extremely rare; it is usually vertical. The author's patient was a girl of six months, with the history that the right eye began twitching from side to side rapidly when she was two months old. The rate was about 250 per minute. Closing the right eye produced no nystagmus in the left, and closure of the left eye had no effect on the right. The child had influenza when examined; she was better in three weeks, but the nystagmus was unaltered. A month later homatropine drops were used for examination purposes, after which there was a slowing of the eye movements. A few days later the mother

reported that the movements had ceased. According to Herman, this condition appears commonly in the first year and generally in the dark months of the year. This child came of poor country stock and from a home in which illumination was insufficient. The author compares this nystagmus with that found in coal-miners and with that produced experimentally in dogs. The prognosis is good and the treatment is to give the child more light. A mydriatic should be instilled.

OTO-RHINO-LARYNGOLOGY.

Chronic Deafness in Children.

J. A. KEEN (*The Journal of Laryngology and Otology*, December, 1934) describes clinical observations on chronic deafness in children. He gives the clinical histories of thirty-two children suffering from chronic deafness. The patients were under observation for a varying number of years, the majority of them for a seven-year period. The records of the hearing capacity are given in the form of graphs. The patients have been divided into three main groups: those with chronic suppurative *otitis media*, chronic middle ear catarrh, and otosclerosis. In addition to the usual methods of treatment, rather more than half of the children had courses of treatment by the Zund-Burguet electrophonoid method ("treated cases", while the remainder did not have this special treatment ("controls"). The author holds that the differential diagnosis and classification of chronic deafness are unsatisfactory, and there is much difference of opinion among otologists. The classical symptoms and signs are of little help and the growing tendency of otologists to make a diagnosis of otosclerosis is discussed. The distances for the conversation voice and the whisper are still the most commonly used hearing test. Provided certain precautions are taken, they remain the most satisfactory method of testing the hearing capacity. There was no evidence in the series of cases that weather conditions had any influence on the hearing. The author has discussed the significance of auditory intelligence and auditory acuity, and suggests that too much importance is often attributed to auditory intelligence. The simple number tests which he has used are a true measure of the auditory acuity, and for practical purposes auditory intelligence does not come into play. The relation between hearing and the appearances of the tympanic membranes is considered. In chronic deafness the cases with normal tympanic membranes are often the worst as regards interference with hearing and prognosis. Pathological changes on the inner wall of the tympanic cavity are probably more important in explaining the deafness than lesions of the tympanic membrane and ossicles. Considerable fluctuations in the hearing capacity are found and in consequence it is difficult to give an accurate prognosis. Chronic deafness in children is by no means always progressive. At the end of the observation period the hearing was better in forty-two, worse in nineteen and unchanged in three instances. Lastly, the author has considered possible methods of treatment and has explained the origin of the Zund-Burguet electrophonoid method. Previous attempts to stimulate the hearing by auditory exercises date back to Urbantschitsch (1895). The electrophonoid treatment is suitable for children and is an easy method to apply. It appears to be particularly successful in cases of deafness remaining after chronic otorrhœa and in chronic middle ear catarrh.

Reinfection of the Wound following Mastoidectomy.

ALFRED A. SCHWARTZ (*Archives of Otolaryngology*, January, 1935) discusses reinfection of the wound after mastoidectomy and gives the histories of five cases. He points out that the desired end-results after simple mastoidectomy are complete healing of the wound and a dry middle ear. The healed cavity first fills with firm granulations, which soon become converted into connective tissue. Into this, mainly from the periosteum, grows new bone. The amount of new bone and the rapidity of its growth vary greatly. There have been cases in which, many years after the primary operation, a secondary operation has shown little new bone; in others what appeared to be a practically normal mastoid was found, with small cells and even normal anatomical outlines. As a rule the regeneration of bone is somewhere between these two types, and the mastoid process rarely recovers its original normal appearance. The new bone forms a hard shelf which covers a cavity about the antrum. This cavity is filled with firm connective tissue which does not change. Reinfection of the wound following mastoidectomy is frequent. In the vast majority of cases a small incision through the lower end of the wound is sufficient to drain the abscess, and complete recovery ensues. Complete mastoidectomy should be considered only when there are signs or symptoms of threatening complications, or when there is evidence of an incomplete surgical procedure at the original operation. Many cases of reinfection of the wound clear up without incision if drainage through the aditus and middle ear is sufficient.

Tuberculosis of the Larynx.

HENRY P. SCHUGT (*Archives of Otolaryngology*, February, 1935) discusses the diagnosis and treatment of tuberculosis of the larynx and states that this condition is curable in patients with good general resistance. Close cooperation with the general practitioner and the surgeon is necessary before local treatment of the larynx is started. Direct laryngoscopy for the correct diagnosis is preferable to indirect examination with a mirror. Emphasis is laid on the importance of the laryngeal ventricle in incipient cases of laryngeal tuberculosis. A preliminary puncture with the galvano-cautery tip is valuable in ascertaining the healing power of the patient. Electro-cautery is of great value in the routine treatment of the tuberculous larynx. The curative paralysis in one-sided involvement is a valuable method, although little is known of it. In laryngeal obstruction from tuberculous lesions tracheotomy should be avoided and replaced by direct intralaryngeal operations. In treating dysphagia, blocking of the superior laryngeal nerve is the most suitable method. Ordinarily this is done from the outside. The nerve can also be blocked from the inside through the pyriform sinus. Resection of the nerve should be limited to patients in good general condition. In cases of pharyngeal tuberculosis severe pain can be dealt with by blocking the pharyngeal nerve plexus. Small lesions in the mouth can be excised and treated locally with success. In advanced cases of lingual tuberculosis with severe pain conservative treatment may be useless. The pain in this area can be destroyed either by blocking the lingual nerve with alcohol injected intraorally or by resecting the nerve from the outside.

Congenital Web of the Larynx.

GABRIEL TUCKER (*Archives of Otolaryngology*, February, 1935) reports a case of congenital web of the larynx in a girl aged twenty years. The character of the web was unusual, the posterior half being thin and transparent and the anterior portion showing fusion of the margins of the cords, with a very thick subglottic extension. The condition responded well to the method of treatment used, namely, incision, the local application of compound tincture of benzoin to the freshly cut edges, and the gentle separation of the margins of the cords to prevent adhesions.

Vasomotor Rhinitis.

A. R. HOLLENDER (*Archives of Otolaryngology*, April, 1935) presents a paper with the object of calling attention to a method of ionization which has a pronounced and lasting palliative effect in cases of vasomotor rhinitis. The technique of intranasal ionization is described and the report of five cases is given. It is not claimed that intranasal ionization is a cure for vasomotor rhinitis. However, the author is of the opinion that the palliative effects are so gratifying as to justify all rhinologists in availing themselves of the method in order to afford their patients relief for a comparatively long period.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on July 5, 1935, DR. W. N. ROBERTSON, the President, in the chair.

Maternal Mortality.

DR. KENNETH WILSON read a paper entitled "Notes on Maternal Mortality" (see page 281).

DR. M. ELLIOT SMITH read a paper entitled "Maternal Mortality: Some Practical Points in Prevention" (see page 285).

DR. R. A. KEYS congratulated Dr. Wilson and Dr. Elliot Smith on their papers. While attending the out-patient department at the Rotunda Hospital over a period of six months, he had taken a series of blood pressure estimations in all types of patients, including those who had obvious albumin in their urine. As a general rule he had found subnormal pressures, and in a discussion with Dr. Davidson he had seriously discussed whether pregnancy, the presence of albumin in the urine and a raised blood pressure had any relationship. He stated that in occipito-posterior presentations infants were delivered with the face to the pubis. At the Rotunda Hospital no rotating was done. Dr. Keys was anxious to know what Dr. Wilson and Dr. Elliot Smith thought of the treatment of eclampsia, and quoted the different methods used in Dublin and in Glasgow. He also inquired as to the type of Cæsarean section done and the time chosen to do it.

DR. ALEX. H. MARKS congratulated the speakers on their papers. There had been improvement in the matter of getting patients into decent private hospitals and he thought this would continue. If the patient was being confined at home the relatives could be very annoying, trying to hurry one up; then the nurse might be good or she might be young and inexperienced. In hospital one could do much more with the patient, and hospitals had improved very much. The question of expense also came into the matter. Dr. Marks considered that there was one condition in which Cæsarean section was permissible, and it was of a *primipara* with a central *placenta prævia;* it was fair to the mother and the child. In a *multipara* Cæsarean section was not so essential. With regard to the rise in blood pressure and albumin in the urine Dr. Marks said that it had formerly been his practice to test for albumin only, but recently he had come round to Dr. Elliot Smith's view that the blood pressure was important.

In occipito-posterior cases the head should be rotated if possible, though this could not always be done. Also some patients would deliver themselves with the fœtus in the occipito-posterior position, while numbers of fœtuses were rotated without artificial aid. Obstetrical specialists often made statements which were taken notice of and which were perhaps not correct. One specialist had said that no fœtus presenting a breech with extended legs and arms could be delivered alive, and that an X ray examination should be made, and if the fœtus had extended arms and legs Cæsarean section could be performed. Dr. Marks found this quite wrong, as the child could be delivered. Often these people were holding teaching positions, but they had not always teaching experience.

DR. C. C. MACKELLAR asked whether the figures showing the mortality from embolism were approximately the same for another period of five years; also was the total number of births given in these papers.

DR. D. GIFFORD CROLL had been interested in the papers; Dr. Wilson had said all these deaths were not preventible and he was glad to hear this fact stated. He was interested to see the comparison between the death rates in New South Wales and Queensland, and thought that one explanation that might be offered was that most of the casualties were due to haste; there were in Sydney men with very big practices who could not give the necessary time to maternity cases, and he thought this would explain the higher figures. Dr. Croll was interested to see the comparatively good figures from the Queensland Obstetrical Section. Dr. Wilson had said that there was very little obstetrical training to be obtained, but a practitioner could join up with the Obstetrical Section, and could get discussion and instruction on his cases, and the section was very valuable in this way. He hoped the section would continue and would increase its membership, as it was the nearest thing to post-graduate teaching. Dr. Croll was interested in Dr. Elliot Smith's remarks as to the significance of blood pressure and albumin in the urine. He himself always did both tests and nearly always found that the blood pressure was higher a fortnight or so before albumin appeared in the urine. It was not necessarily "high blood pressure" that was significant, but "increased blood pressure". One should commence taking the blood pressure early in pregnancy.

DR. ALEC CAMERON said that he had found Table IV very interesting, as it was a fairly good answer to the papers and to politicians' remarks, as these figures were sent in by those who were doing general practice in small houses, as it had been done for years; he thought the rate of 3·2‰ was outstanding. He had looked up the figures of different countries for the year 1920; in Victoria the rate had been over 5‰; in the United States of America it was about 1 in 154, in Great Britain about 1 in 210, in Scotland about 6·2‰, and in Edinburgh itself about 8·2‰. It was ridiculous for people to say matters were worse now than they were then; the general figures seemed to be about 5‰. The efforts of the Obstetrical Section were quite encouraging.

He did not think one should say never to do Cæsarean section in eclampsia, as he had known it to be successful. It was very difficult to give practical obstetrical training and to get improvement; longer training before starting in practice was desirable. An effort should be made to try to develop and to improve ways of relieving the pains of labour, and so of lessening shock, and one should not be urged into hastening matters. One of the most important ways of improving midwifery was along these lines.

DR. A. C. F. HALFORD said he did not wish to speak in a deprecating fashion concerning the papers read. He listened to them with interest and offered his thanks to the authors, but he found the position outlined very much the same as ten years ago. It was stated then as now that what was needed were better teaching in obstetrics for obstetrician and nurse, more maternity beds in large costly institutions, and a wider appeal to pre-natal care. In spite of the more intense application of these measures, maternal mortality had increased, as he predicted would be the case ten years ago. He referred only to the factor of sepsis. Dr. Marks had stated that it was better to get the patients into hospitals; he did not say what kind. In the morning papers it was stated that the small private hospitals were a menace, and the Home Secretary said he was going to close them up. Dr. Halford claimed that the best results were obtained in domiciliary midwifery and in private hospitals, and the worst in large, well equipped public hospitals. The Obstetrical Section justly patted itself on the back on its good results, but there were organizations in Australia and in the Old Country in which the mortality was in the region of 1‰. He thought they should try to approach these figures and should not say too much until they did. They should strike out on some new line. There had been leaders, specialists in the profession, who had laid down rules of procedure and decided that the general practitioner was better out of the way. He thought the general practitioner was entitled to keep his cases. How was he to build up a practice and become the family doctor if midwifery was denied him? Ninety-eight *per centum* of women could and should be confined in their own homes under existing conditions, if they preferred to remain there. Some certainly had to be removed to hospital. Doctors must remember that they had built up their practices

largely on the good name and recommendation of nurses, particularly the proprietors of private hospitals in suburbs and country who had invested their savings and worked up worthy connexions. It was unjustifiable and cruel to turn upon them now. The Chairman of the Medical Research Council had stated two years ago that women should stay in their homes, as the hospitals were dangerous places. This showed the state of panic which existed and which extended to the expectant mother. Dr. Halford had hoped to hear of a new line of attack to get rid of a state so deplorable.

DR. J. LOCKHART GIBSON inquired whether, when one had detected a rising blood pressure, one could prevent the occurrence of albumin? He agreed that politicians should not be allowed the full control of medical matters.

DR. J. G. WAGNER congratulated and thanked the speakers. He asked Dr. Wilson had he the details of the situations in which the fatalities mentioned in the notes of the Obstetrical Section had occurred. Were these at home or in hospital? He mentioned that there were practitioners carrying on large midwifery practices without ante-natal care who were very skilled at getting out of tight corners. The best way to deal with the situation was to give the women some idea, without frightening them, of what they were entitled to. The difficulties of labour cropped up so rarely that one might meet them only very infrequently and then be unsuccessful.

DR. W. N. ROBERTSON mentioned the conditions prevailing in Edinburgh when he began his obstetrical work. One had no idea of the poverty in those large cities and could not expect to see results as in Australia, where there were good food and plenty of light and air. Also in Australia rickets did not occur as they did in those slums. So the figures were hardly comparable.

Dr. Wilson, in reply to Dr. Keys, said that most patients with disproportion had a trial of labour before Cæsarean section was done. He did the classical operation and had found no cause to regret it; he had not found it risky for the patient.

Dr. Wilson agreed with Dr. Marks as to the effect of relatives on the practitioner when the patient was confined at home. Eardley Holland said that the method of reducing the mortality rate below 3‰ was to increase the skill of the obstetrician. By increased post-graduate training for the practitioner after gaining experience in hospitals where they confined abnormal cases themselves, they would develop an obstetrical conscience and then they would do what they thought best without worrying about the relatives.

Dr. Wilson told Dr. McKellar that he did not know the total number of births; he only had the comparative numbers. The rates were worked out by the statistician. He entirely agreed with Dr. Croll as to the valuable post-graduate training which was obtained by being an active member of the Obstetrical Section, and thought that membership in this section would help to reduce the maternal mortality rate. Dr. Wilson told Dr. Halford that he had not put forth anything new as a solution, as he was hoping for more from the discussion. If matters went on for some time as they were, and this they must, and if organized effort were made to improve the existing conditions, the mortality rate would be reduced. The mortality rate was always higher in well equipped hospitals, as the patient in the home, if she were not doing well, was often sent into the well equipped hospital. In reply to Dr. Wagner, Dr. Wilson said that the details of the fatalities were in the records; he could not say off-hand whether the patients were confined at home or in hospital. Those successful practitioners who did no ante-natal work had probably in their early years, while becoming experienced, had a high mortality rate, which had lessened with the years; this was where post-graduate practice would come in, lessening the practitioner's rate and the general rate.

Dr. Elliot Smith, in reply to Dr. Keys, said he had found in his patients that a rise in blood pressure occurred with albumin in the urine and often before albuminuria. Brown, of London, had found in 75% of cases that a rise in blood pressure was the first sign of toxæmia.

In occipito-posterior cases the necessity for manual rotation of the head had to be decided on its merits; he could not lay down a definite rule; in his paper he was speaking of rotation followed by too early delivery. The treatment of eclampsia could not be reduced to a few sentences; he treated it as a toxæmia and left the pregnancy alone for the time—that was his general rule. Dr. Elliot Smith said he always performed the classical Cæsarean section, if necessary after a trial of labour, and here again the case must be judged on its merits. He had heard of the lower segment operation being done when the patient was not in labour; but then no lower segment had been formed and there was no advantage in the lower transverse incision. He was glad to hear Dr. Marks agree as to the difficulty of dealing with relatives. If Cæsarean section were done in a case of central *placenta prævia*, he considered it was done in the interests of the child and was not justifiable for the mother; one could always deliver the child *per vias naturales* with safety to the mother. It was only justifiable, in his opinion, in an elderly *primipara* with a central *placenta prævia* and not in labour. If the child were dead, the section would not be justifiable.

Dr. Elliot Smith agreed with Dr. Croll that not all deaths were preventible, but they must be discussed. He did not like the idea of Cæsarean section for eclampsia, as quoted by Dr. Cameron. The published statistics showed the greater dangers of surgical treatment. Conservative methods still showed easily the best results.

Dr. Elliot Smith told Dr. Halford that his paper was producing nothing new; indeed it was very difficult to produce anything new. If all the minor points mentioned were noted and followed by all medical practitioners, the maternal mortality would be reduced. The waving of any magic wand would never alter it. He was in favour of normal patients being confined at home, but did think that the medical practitioner should be given more time to attend them, possibly by group practice or some such means. The public hospital was not necessarily a short road to the grave, as was shown by comparing the results of the Lady Bowen Hospital and the figures of Queensland and New South Wales. The smaller, poorly staffed maternity homes were a definite danger, and a patient was safer in her own home. Good hospitals with separate labour wards and full equipment or none!

Dr. Elliot Smith told Dr. Gibson that one could attempt to prevent eclampsia and generally succeeded; calcium gluconate seemed to help. If albumin was present in the urine and the blood pressure was raised and the outlook for the patient was bad, an induction could be done, and this, in his opinion, should always be surgical.

Dr. Elliot Smith agreed with Dr. Wagner that ante-natal care was absolutely necessary. He thought it was the many small practical details mentioned in the paper which constituted the attack on maternal mortality. People were now getting educated on the subject and had some idea of what they were entitled to.

ANNUAL MEETING, MELBOURNE, 1935.

Registration.

All members attending the forthcoming annual meeting of the British Medical Association must register their names at the registration room in the Anatomy School at the University of Melbourne at the commencement of the meeting in order to receive a member's card, the programme of the meeting and tickets for various functions. Notification to the office of intention to attend the meeting does not absolve any member from the need of registration.

In order to facilitate the registration of members, it is proposed to open the registration room on Friday, September 6, from 9 a.m. to 5 p.m., and on Saturday, September 7, from 9 a.m. to 12 noon. All members in the

metropolitan area are urged to register at these times if possible, but no tickets will be available until Monday, September 9.

Ladies' Social Club.

The wives of all members attending the meeting must register their names at the Ladies' Social Club, Oriental Hotel, Collins Street, Melbourne, at the commencement of the meeting, in order to receive a badge, the Ladies' Handbook and tickets for various social functions.

In order to facilitate the work of registration, the Ladies' Social Club will open on Saturday, September 7, from 9.30 a.m. to 12 noon. The wives of all members in the metropolitan area are urged to register at this time, but no tickets will be available until Monday, September 9.

Victorian Branch Ball.

Those members of the Victorian Branch who wish to act as hosts at the Branch Ball, to be held on September 12, 1935, and who have not already notified the Medical Secretary, 426, Albert Street, East Melbourne, are requested to do so as soon as possible.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Twohig, Francis Vincent, M.B., B.S., 1935 (Univ. Sydney), 19, Annandale Street, Annandale.

Correspondence.

ETHYL CHLORIDE IN ANÆSTHESIA.

SIR: There has been considerable dispute recently about the danger of ethyl chloride being administered preliminary to ether for general anæsthesia.

I have taught this method for twenty years, and I know of several hundred medical men who carry it out without any complications. I myself have used this method in, I should say, twelve or fifteen thousand anæsthetic cases. At very odd times I have had trouble, and in every case when this happened my technique was to blame. As soon as I corrected it, I had no more trouble.

The main points in using this method are as follows:

Use coarse cheese cloth gauze on the face mask, about six layers; never use finely woven gauze, and on no account use a thick pad of gauze on the mask. A good plan is to rumple the coarse gauze between the hands and then try to blow through it. If the air passes freely through, without any obstruction, the gauze is fit for use. If, however, obstruction is felt to the breathing, the pad should be discarded or made thinner.

The other point to note is never to give too much ethyl chloride. Spray on a few drops of ethyl chloride and ask the patient whether it is too strong. As soon as that has evaporated, that is, in about four seconds, spray on some more, still quietly questioning the patient. Keep on doing this until the patient speaks in a drunken way, or only mumbles, and then stop administering the ethyl chloride, and, in a few seconds' time, pour on a thin stream of ether.

If suitable gauze is used, that is, gauze which does not absorb too much ethyl chloride and keep it locked up, and the subsequent method is adhered to, I am quite certain that those practitioners who have had trouble with this sequence will not have any further anxiety. They should remember that the anæsthesia from ethyl chloride continues to deepen for about eighteen seconds after the administration of this drug has been stopped.

Yours, etc.,

MARK C. LIDWILL.

135, Macquarie Street,
Sydney,
August 19, 1935.

Obituary.

DIXIE PAUMIER CLEMENT.

WE are indebted to Dr. G. W. Barker for the following account of the life of the late Dr. Dixie Paumier Clement.

Dixie Paumier Clement was the elder son of Mildmay Charlton Thomas Clement, M.A.I.E., M.I.C.E. He was born at Dungamoor, County Tyrone, on December 23, 1879, and was educated at St. Faughnan's College, Rossearbery, County Cork. He came to Western Australia with his family at the age of sixteen years and he went straight to the goldfields, where later on he was one of the four who discovered the Lancefield mine, on which he worked until 1902, when he came to Perth and took his matriculation, and one year later left for Dublin, where he studied and took his degrees: B.A., M.B., B.Ch., B.A.O., at Trinity College, Dublin, and L.M., Rotunda, Dublin, and returned to Western Australia towards the end of 1908.

One week after his arrival he did locum tenency work at Pingelly for six weeks, and on his return to Perth he went into partnership with the late Dr. Athelstan Saw and commenced practice at his residence at the corner of Hay and Colin Streets in December, 1908. He married Miss Ethel Burt on September 1, 1909.

During his years of practice he held many honorary positions, namely, honorary physician to the Perth Hospital, honorary medical officer to the King Edward Memorial Hospital for Women from its formation. He also attended in an honorary capacity the Home of Peace for the Dying and Incurable, the Home of the Good Shepherd, and, after the death of the late Dr. Merryweather, St. Brigid's Convent, West Perth. After the death of Dr. Saw he took his place as one of the trustees of the Padbury bequest. He was also a member of the Dental Board for nine years, Medical Board for five years, Australasian Trained Nurses' Association Council, and took a very active interest in the affairs of the Western Australian Branch of the British Medical Association. He was on the Branch Council for eighteen years, during which time he was Honorary Secretary for four years, and President in 1924. He was a great upholder of the traditions of the profession, and, as one member has truly expressed: "He was a wise counsellor and a good friend, both to the Association collectively and to its members individually."

Dixie Clement will be sadly missed, not only by the medical men of Perth, but of the whole of Western Australia. His principal work, and work which he loved, was obstetrics. He was at every man's beck and call whenever there was a difficult case, and he would be at the bedside in a very few minutes after receiving the call, with a kindly word for the patient and also for the doctor who had called him. It was a well known saying that: "Dixie will never let you down."

For relaxation he took up golf and was one of the foremost amateurs for many years in this State. He was a member of the Perth Club and a foundation member of the Lake Karrinyup Club, and at different times was captain and president of both clubs. Early this year he gave up competitive golf and played only a few games, as he was feeling the strain and he felt that he must give all his time to his practice. This devotion to duty was no doubt the cause of his death, for, since he landed in Western Australia at the age of sixteen years, he had never taken a holiday longer than one month and had never left the State since 1908.

Shortly before his death, while attending a fellow practitioner's funeral, he passed the remark that it was an inhuman custom to have people showing their harassed feelings at the graveside, and he intended to make certain that it would not occur at his. He showed that he meant it, for he had the very simplest of funerals unattended, but some of his oldest male friends and the doctors attended at his graveside. He is survived by a widow, four daughters and two sons.

Dr. D. D. Paton writes:

Dixie Clement was probably not well known in the eastern States, but in Western Australia no medical practitioner was more widely known and more highly respected. That death has removed him can scarcely be fully realized, for he was never sick, was always about his work, with a ready salutation on his lips for all.

When he recovered his faculties after a few hours' loss of them in the forenoon of Monday, July 15, his medical attendants and all his friends hoped that he had but received a kindly warning that for too many years he had been overworking. However, some hours later the full force of the attack developed and it became evident there was being planned a close to his long day of service. No one wanted to see his splendid vigour lamed, nor his eager heart chafed by an inability to enter into the high privilege of living, yet, when the silence was already falling, the desire to keep him back, where he had endeared himself to all, was at its strongest. However, at the end nothing was left but for the last gathering of his sorrowing friends that had joined in the benediction of the Church to turn back into a world made poorer for his absence.

The early years of his life after his father's demise had not been too easy, but no stouter heart than his ever battled more cheerfully on towards a chosen goal. His professional skill and contemporary esteem prove he arrived at it, and patients throughout the length and breadth of the State would testify to it with ready voice.

As he made his life, he added to his store of inherited right-mindedness a treasury of disciplined thought and restraint, so that, although he never thrust himself forward, one found he was always ready with friendly advice when it was asked for, and was tactful in the giving of it.

His nature was essentially sunny and radiated happiness wherever he went, but underneath was concealed a seriousness and self-possession that brought with his friendliness a sense of sincerity and uprightness quite incompatible with unworthy motives or jealousies.

I believe no one was so open to approach from the younger men, and if he had held a post in a teaching medical school he would have proved by example and precept a powerful influence for good amongst students, for he was thorough in work and sport, entering into both spheres with zest. Short holidays for fishing, golfing days made as regular as they could be made in a busy practice, provided his recreation, and so good a golfer was he at one time that his growing family could not understand their father returning without a trophy on all big occasions.

Looking back over his life, one can see how well equipped he was to become a successful medical man. Nature had endowed him with a loving, sympathetic, generous heart, and Dublin gave him the fullest training it could in obstetrics, which was the branch of medicine he specially selected to study. He was physically strong and bore his years of broken nights apparently easily, for he was always ready at any hour to respond to any genuine call. The maternity hospitals gave him freely of their difficulties, and he greeted them with a smile. Indeed much of the present development of the King Edward Memorial Maternity Hospital and the record it holds for the lowest maternity mortality are largely due to him, and it is pleasing to know his name will be perpetually connected with the institution, for the new obstetric ward will bear his name. That is a tribute that might have thrilled him: a certificate issued to posterity that, man and obstetrician, his generation gave him thanks that he had lived. No one would have thought of praising him to his face, beyond a few terse words, for he would have been off before more followed, but, now that he has gone, his merits can be told out of the fullness of one's affection and esteem. All his brethren miss him sadly and so may offer humbly to his widow and family, who have suffered the irreparable bereavement, the tribute of deepest sympathy. Mrs. Clement was a wonderful partner in all his ambitions and ideals, and her sterling character will insure that these ideals will be handed on to the family just as if Dixie were there to communicate them himself.

Dr. Kasner Moss writes:

By the death of Dixie Paumier Clement on July 25 from a cerebral thrombosis Perth lost its foremost general practitioner, and the medical profession its guiding star in obstetrics and ethics.

Dixie (as we all called him) was only fifty-five years old and was universally beloved by the profession. There is not a general practitioner in Perth who has not at some time called on him for help in an obstetric case. Neither time, nor weather, nor money, nor convenience ever entered into his calculations when he heard the call of a brother in distress. He would arrive as fast as his car could bring him, and his cheery tenor voice and smiling optimistic face, crowned by his tawny hair, would bring comfort to the doctor and deliverance to the mother.

His fame as an obstetrician spread the length and breadth of Western Australia. It was his teaching and practice that brought the obstetrics of the King Edward Memorial Hospital to its present high standard.

His capacity for work was enormous. Three confinements in twenty-four hours with anæsthetics sandwiched between (he was an expert anæsthetist) and a long list of visits did not seem to upset him. He has spent the night out of bed at confinements and the next day played off for a golf championship.

It must not be assumed that he had a calm disposition or a docile temperament. Far from it. Like all sanguine men, he was impulsive and even hot-tempered.

He worshipped the truth, and woe betide anyone he detected in a dishonest act. I know of one case, where, having sheeted home ethical dishonesty to a fellow practitioner, he took him to task and told him with a directness that few could equal, exactly what his opinion of him was. Yet within a few months Dixie assisted him in a Cæsarean section, his viewpoint being that, having corrected the man and purged him of sin, the welfare of the patient was the only thing to consider.

He was at the time of his death honorary doctor to the Home of Peace for the Dying and Incurable, to the Home of the Good Shepherd, and to the Brigidine Convent of Perth.

I have been in general practice for about thirty years and I have never met a medical man whom I have admired so much as Dixie Clement. His very faults, stubbornness and bluntness, became virtues in him, by reason of the way he used them. His generosity to all who were sick was almost unbelievable. Only two days before his last illness I called him in to deliver a transverse presentation, as I had a carbuncle on my wrist. I can see him now come smiling into the room with: "What's the trouble, old dear?" He performed a difficult version and delivered a ten pound baby that was dead before I had arrived. I posted him a cheque, and the next day he said to me: "Shall I tear that cheque up or post it back?" On my protesting, his only remark was: "You were sick. Wouldn't you do the same for me?" That was the way Dixie Clement always acted.

And now his tenor voice is silenced for ever, his weathered face, with his cheery smile, has passed from us, and we have only left a shining memory of Dixie Clement, doctor, man and friend.

"There be some, thou knowest, who work on slowly and steadily until nightfall, and some there be who work as though they were giants, and with such hearty goodwill that by noontide their appointed work is done, and their Master bids them rest from heat and burden of the day."

CHARLES HORSFALL ARMITAGE.

WE regret to announce the death of Dr. Charles Horsfall Armitage, which occurred on August 20, 1935, at Mosman, New South Wales.

A MASONIC LUNCHEON.

THE Victorian members of the British Medical Association who are Freemasons desire to invite all visiting Masonic members of the Association to a luncheon to be held on Tuesday, September 10, at 1 p.m., at the Freemasons' Hall, 25, Collins Street, Melbourne. All medical brethren desirous of attending are requested to signify their intention of so doing by filling in immediately the card provided when they register in connexion with the annual meeting of the Association at the University of Melbourne.

A WARNING NOTICE.

THE Medical Section of the Victorian Branch of the British Medical Association writes that a female morphine addict is endeavouring to obtain prescriptions for supplies of morphine from doctors in Melbourne and the suburbs. She alleges that she is a nurse and requires the drug for a patient coming from the country, and goes so far as to arrange hospital accommodation for the fictitious patient.

Diary for the Month.

SEPT. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 3.—Tasmanian Branch, B.M.A.: Council.
SEPT. 3.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 4.—Western Australian Branch, B.M.A.: Council.
SEPT. 4.—Victorian Branch, B.M.A.: Branch.
SEPT. 5.—South Australian Branch, B.M.A.: Council.
SEPT. 6.—Queensland Branch, B.M.A.: Branch.
SEPT. 9-14.—Annual Meeting of the British Medical Association in Melbourne.
SEPT. 10.—Tasmanian Branch, B.M.A.: Branch.
SEPT. 13.—Queensland Branch, B.M.A.: Council.
SEPT. 17.—Tasmanian Branch, B.M.A.: Council.
SEPT. 18.—Western Australian Branch, B.M.A.: Branch.
SEPT. 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 25.—Victorian Branch, B.M.A.: Council.
SEPT. 26.—South Australian Branch, B.M.A.: Branch.
SEPT. 26.—New South Wales Branch, B.M.A.: Branch.
SEPT. 27.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. H. J. Eizenberg (B.M.A.) has been appointed Honorary Ear, Nose and Throat Surgeon, Liverpool State Hospital and Home, Office of the Director-General of Public Health, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi, xvii, xix.

ALFRED HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.
BLAIR ATHOL DISTRICT HOSPITAL, QUEENSLAND: Medical Officer.
BOULIA HOSPITAL, BOULIA, QUEENSLAND: Medical Officer.
FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Resident Junior Medical Officer.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
ROYAL AUSTRALIAN NAVY: Medical Officers.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Senior Resident Medical Officer.
THE PRINCE HENRY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Radiologist.
WELLINGTON HOSPITAL BOARD, WELLINGTON, NEW ZEALAND: Tuberculosis Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.